AD-A061 674 AIR FORCE INST OF TECH WRIGHT-PATTERSON AFB OHIO F/G 6/20
LEAD - AN EPIDEMOLOGICAL LOOK AT POTENTIAL SOURCES OF CONTAMINA--ETC(U)
1978 W TAYLOR
UNCLASSIFIED AFIT-CI-79-60T NL

1 OF 1
AD A061674

END
DATE FILMED
2-79
DDC

UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE *(When Data Entered)*

AFIT – REPORT DOCUMENTATION PAGE	READ INSTRUCTIONS BEFORE COMPLETING FORM
1. REPORT NUMBER: CI-79-60T	2. GOVT ACCESSION NO. / 3. RECIPIENT'S CATALOG NUMBER
4. TITLE *(and Subtitle)*: Lead - An Epidemological Look at Potential Sources of Contamination	5. TYPE OF REPORT & PERIOD COVERED: Thesis
	6. PERFORMING ORG. REPORT NUMBER
7. AUTHOR(s): Wiley Taylor (Master's Thesis)	8. CONTRACT OR GRANT NUMBER(s)
9. PERFORMING ORGANIZATION NAME AND ADDRESS: AFIT Student at the University of Arizona	10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS
11. CONTROLLING OFFICE NAME AND ADDRESS: AFIT/CI, WPAFB OH 45433 (92p.)	12. REPORT DATE: 1978
	13. NUMBER OF PAGES: 85
14. MONITORING AGENCY NAME & ADDRESS *(if different from Controlling Office)*	15. SECURITY CLASS. *(of this report)*: UNCLASSIFIED
	15a. DECLASSIFICATION/DOWNGRADING SCHEDULE

LEVEL II

16. DISTRIBUTION STATEMENT *(of this Report)*

Approved for Public Release, Distribution Unlimited

DDC RELEASED NOV 29 1978 F

17. DISTRIBUTION STATEMENT *(of the abstract entered in Block 20, if different from Report)*

18. SUPPLEMENTARY NOTES

APPROVED FOR PUBLIC RELEASE AFR 190-17.

JOSEPH P. HIPPS, Major, USAF
Director of Information, AFIT

NOV 9 1978

19. KEY WORDS *(Continue on reverse side if necessary and identify by block number)*

20. ABSTRACT *(Continue on reverse side if necessary and identify by block number)*

AD A061 674

DDC FILE COPY

DD FORM 1 JAN 73 1473 EDITION OF 1 NOV 65 IS OBSOLETE

012 200

UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE *(When Data Entered)*

79-60T

LEAD--AN EPIDEMOLOGICAL LOOK AT POTENTIAL SOURCES OF CONTAMINATION

A Thesis submitted to the

Division of Graduate Studies
of the University of Cincinnati

in partial fulfillment of the
requirements for the degree of

MASTER OF SCIENCE

in the Department of Environmental Health
of the Graduate School of the College of Medicine

1976

by

Wiley Taylor
B.S., University of Arizona, 1972

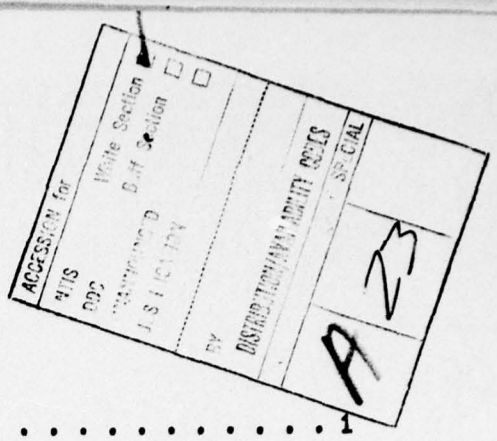


TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

79-6DT

THESIS ABSTRACT

This thesis is a critical review of the literature on the occurance of lead, lead products, and their intrarelationship to the epidemiology of lead poisoning.

Lead has always been present in the soil, in water, and in air, following the burning of wood and coal; also in plants, both edible and inedible. Lead in soil ranges from 2-200 ppm and averages 16 ppm. Cholak, Schafer, and Sterling found the lead content of soil in Urban Cincinnati to be up to 360 ppm. In natural bodies of water, the concentration is much lower - between 0.001 and 0.01 ppm. Throughout recorded history, lead has been well known and widely used by mankind. The prehistoric metal was used for glazing pottery by the early Egyptians dating back to 7000-5000 B.C. In the United States, some lead was mined in Virginia in 1621 and the discovery of lead in the Mississippi Valley was reported in 1960. In 1763, the lead industry started production on a permanent basis. Since that time, lead has continued to be mined and used on an escalating basis. Mine production in the United States in 1974, was 669 thousand tons, while the domestic consumption was 1550 thousand tons. Since 1763, when the lead industry began in this country, 94 billion pounds of lead (this total excluded all lead that has been recycled), have been consumed in products that are now in our environment.

The hazards of lead toxicity in the mining and manufacturing of lead when lead dust and particularly lead fumes are encountered , are well known and monitored. However, there may be many additional sources of lead in the environment that have the potential for adding to the total

body burden of lead as evidenced by increased biological lead levels. It is imperative that those personnel engaged in Industrial Medicine be cognizant of these and be able to evaluate their potential effect on the industrial worker. This thesis covers the history of the Lead Industry within the United States, the sources of lead used in commercial products and sources of lead in the environment, and a review of the epidemiological lead poisoning cases appearing in recent literature.

I. INTRODUCTION

Lead is one of the first metals used by ancient man. It occurs everywhere in man's environment, in his food, in his utensils, in his home and in almost everything which comes into intimate contact with his tissues (1). In all probability, minute quantities of lead enter his system while he is still nourished in the placenta. Lead remains in his tissues throughout maturation and until death. Barring any massive accumulation of lead within the system or any greatly increased lead consumption above "normal," the lead appears to be a benign contaminant within the biologic system. In the "normal" range of concentration lead appears to play neither a beneficial nor a detrimental role. However, when the normal range is greatly exceeded and there is a massive body lead burden, we get into what is termed lead intoxication.

The poisonous properties of lead were discovered early. The ancient Egyptians are said to have used lead for a homicidal poison. Inadvertently, the Egyptians poisoned themselves by using lead containers to store their wine. At the same time, their slaves fell victim to occupational lead poisoning as they mined, smelted and manufactured lead products for their masters. As early as the Fourth Century, B.C., lead toxicity in the mining industry was recognized and recorded by Hippocrates (2).

Lead has perhaps been studied more than any other metal on Earth. Certainly more has been written on the properties of lead and its compounds and their effects on the biologic system than any other metal. In fact, there have been so many studies and such voluminous amounts of printed words on the subject of lead that it staggers the imagination. The amount

of knowledge, unfortunately, does not correlate even closely with the volume of the literary output on the subject (1). Many studies, at least to the beginning student, seem almost irrelevant, and tend to lose sight of their objectives. Other studies are confusing and there are apparent contradictions among the experts. Yet, much about lead and the biologic system remains unknown. For example, it is not known whether some quantity of lead is essential or even physiologically useful to the living organism. Lead is ubiquitous, so ingrained in living tissue that it is nearly impossible to study cell life in the absence of lead.

For the average health scientist to wade through, examine and interpret the current literature would require great quantities of valuable time and unimaginable fortitude and he would probably end up confused and in despair. So much has been said about lead, so many hypotheses on the effects of lead have been postulated, and so many opinions have been offered that a search of the literature could lead to bewilderment and confusion. The world renown expert on lead, Robert A. Kehoe, has stated that laymen know little of the poisonous properties of lead and that few physicians outside of industry have any experience that qualifies them to diagnose or treat lead poisoning (3). It has been assumed that many cases of lead poisoning, especially those occurring in very young children or among adults who have no obvious exposure to lead, go unrecognized.

It is the purpose of this paper to glean from the prestigious annals of science a simplified thesis on lead and its compounds. It is hoped that by enumerating lead manufacturing processes and products, this will give the health scientist insight as to where lead hazards could exist. This paper will discuss lead in the environment and some of the processes

which have added to that lead. Its aim is to cover the most recently reported cases of lead poisoning and to correlate the lead poisonings to lead products or processes. To attempt to write a comprehensive critical review of the mass of literature on the biological effects of lead and its products would be foolhardy and presumptuous; rather, this paper will offer a skeleton framework of pertinent information on the lead industry, lead in the environment, and lead poisoning.

II. LEAD PRODUCTION AND UTILIZATION IN THE UNITED STATES

A. Background

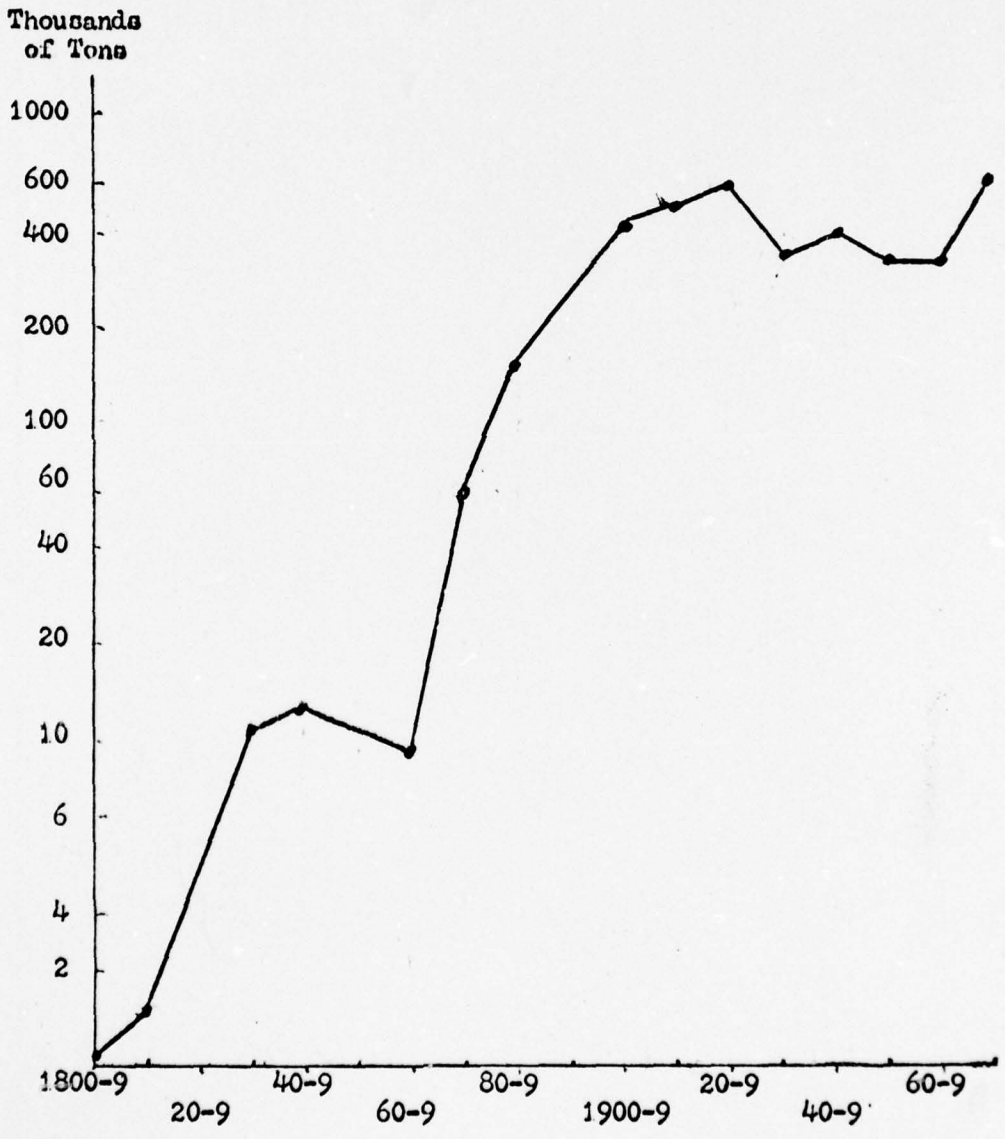
Throughout recorded history lead has been well known and widely used by mankind. Lead was among the first metals known to the early Egyptians, Hebrews and Phoenicians and many of the ancient applications have persisted through the centuries. The prehistoric metal was thought to have been used for glazing pottery by the early Egyptians dating back to 7000-5000 B.C. The earliest known specimen of lead, a figure from the area of the Dardanelles on the site of an ancient city called Abydos, dates from 3000 B.C. Old lead pipes have been found in Egypt and the hanging gardens of Babylon were said to have been floored with soldered sheets of lead (4).

In the United States, some lead was mined in Virginia as early as 1621 and the discovery of lead in the Mississippi Valley was reported in 1690. In 1763 the lead industry started production on a permanent basis (5). Since that time lead has continued to be mined (Figure 1) and used on an escalating basis (Figure 2 and Table 1). Mine production in the United States in 1974 was 669,000 tons, while the domestic consumption was 1,550,000 tons (6). Although the United States has been the world's leading lead-mining nation from 1929 to 1974, it has required additional sources of lead to meet domestic requirements which have increased from 605,000 tons in 1920 to over 1.5 million tons in 1974. Production from scrap (recycled lead) was first reported in 1907 at 26,000 tons and in 1974 it was up to 632,000 tons, nearly 41 percent of the total consumption. Approximately 3.6 million tons of lead were consumed on a world-wide basis in 1974 (6, 7).

The relative inertness and malleability of lead enhances its usefulness

FIGURE 1: SMELTER PRODUCTION OF LEAD

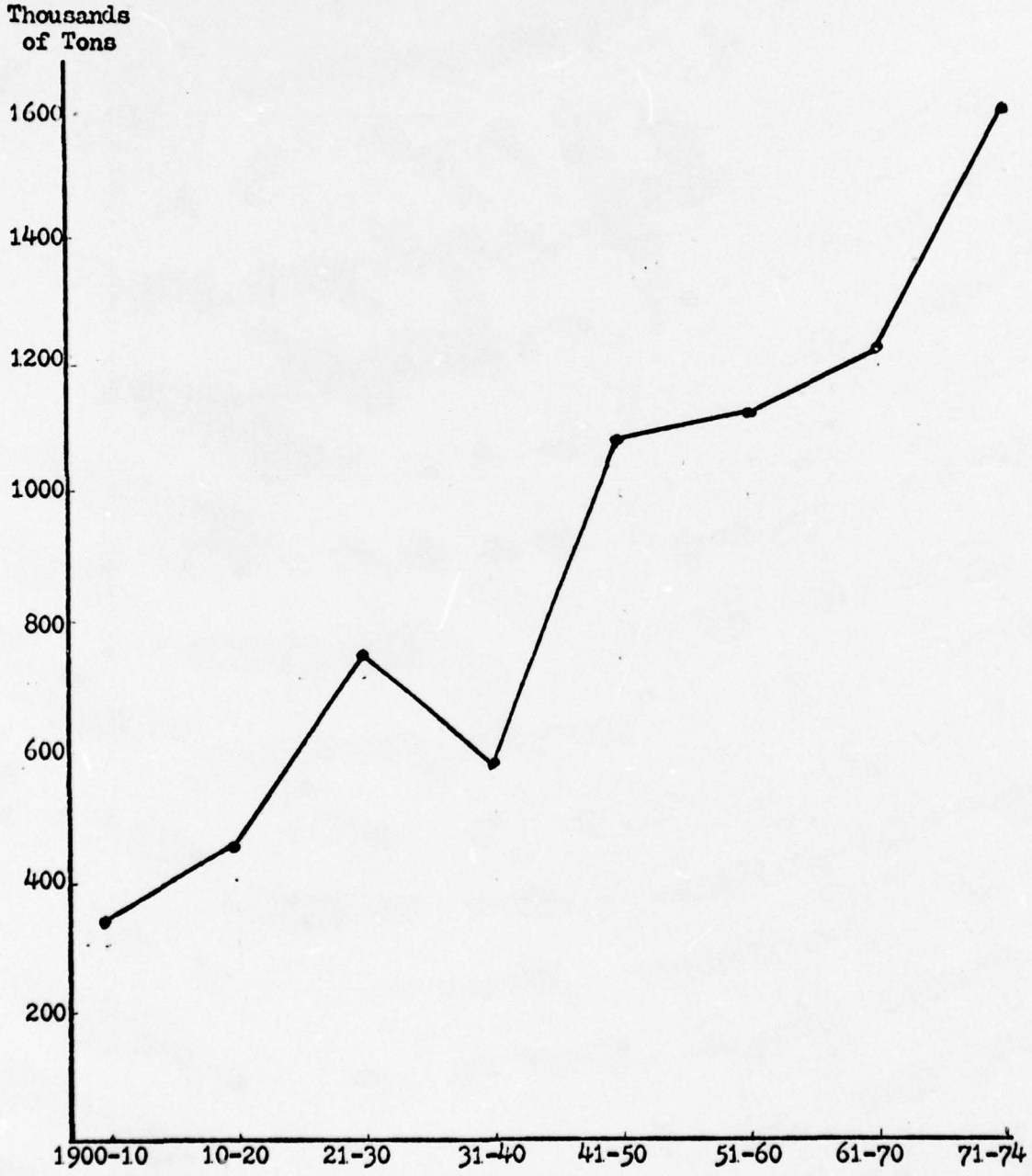
Yearly Output for 10 Year Periods, 1801-1974



From Minerals Yearbook (7)

FIGURE 2: UNITED STATES' CONSUMPTION OF LEAD

Average Yearly Consumption for 10 Year Periods, 1900-1974



From Minerals Yearbook (7)

TABLE 1: UNITED STATES' CONSUMPTION OF LEAD
(In Thousands of Tons)

	1925-30	31-40	41-50	51-60	61-70	71-74	Total
Ammunition	211	349	612	440	648	341	2,601
Bearing Metals	186	125	352	290	194	62	1,209
Brass and Bronze	*	*	*	234	217	83	534
Cable Covering	1,110	669	1,446	799	575	181	4,780
Casting Metals	101	*	*	117	77	35	375
Caulking Lead	176	134	385	598	599	90	1,982
Collapsible Tubes	*	*	*	105	119	20	244
Foil	198	205	116	41	48	18	625
Pipe, Traps and Bends	190	190	382	271	197	73	1,353
Sheet Lead	289	196	358	279	272	96	1,490
Solder	208	189	536	729	704	277	2,643
Storage Batteries	1,138	1,748	3,045	3,590	4,739	3,008	17,268
Terne Metal	23	41	40	19	16	7	146
Type Metal	98	130	239	274	275	83	1,099
Pigment Colors	913	1,163	1,245	1,173	952	279	5,725
White Lead	704	716	502	170	80	12	2,184
Red Lead and Litharge	209	447	743	789	784	310	3,282
Gasoline Additives			728	1,614	2,286	1,067	5,695
Others, Unclassified	460	586	1,307	709	489	286	3,897
TOTAL	5,271	5,770	10,791	11,272	12,407	6,007	51,518

From Minerals Yearbook (7)

*Data not available

to man. It is easily worked and is non-corrosive, making it the ideal metal for a large number and variety of materials and processes. Many of the processes utilizing lead have changed in recent years due to enacted laws and regulations. However, as old processes are deleted, new ones tend to increase the demand for lead. The United States' consumption of lead continues to rise. Trends in the consumption of lead within the United States can be seen in Table 1. Lead is all around us; it starts our cars, until recently was used to keep the knock out of our engines and makes the car bodies sleek and smooth. It was the paint on our houses and remains the corrosive resistant coating on our bridges, is in the glazes on tiles and the porcelain enamel and on aluminum; it is in the finest crystal and optical glass. It shields against radiation. It makes the joints in cast iron pipe and has been the type metal from which we printed (8). Lead radionuclides are useful in geologic dating and documenting the cycling of the metal in the ecosystems (9). Lead is ubiquitous in the environment and is present in small amounts in all living things.

B. United States' Lead Industry

1. General

The United States' lead industry is the largest in the world. It has developed from small, intermittent mining operations producing 1,000 tons per year to 669,000 tons in 1974, which presently accounts for 26.5 percent of the total world output. While United States' mining accounted for 26.5 percent of the world's total output, United States' consumption accounted for 43.3 percent of the world's total, or for 1,550,000 tons.

It is estimated that over 70 million tons of lead have been consumed in the United States since production began in 1763 (7).

The supply of lead to meet domestic requirements is derived from four sources: domestic mine production, imported ore, concentrated imported metal, and domestic secondary metal. In 1974, domestic ore accounted for 88 percent of the 764,000 tons of primary refined and antimonial lead output and imports of ore and bullion for 12 percent. Domestically refined primary lead supplied 44 percent of the 1.74 million-ton apparent demand for lead in 1974; imports of metal provided for seven percent, and secondary lead for 36 percent. The rest came from industry stock reduction and Government releases (6).

2. Primary Sources

Lead is derived from ores varying widely in lead content as well as other metals recovered as co-products or by-products; sources of lead range from the virtually zinc-free lead ores of the old Missouri lead belt through the complex lead-zinc ores of the Western States to the nearly lead-free zinc ores of the Eastern States. Essentially, all ore is mined by subsurface methods, and the ore is beneficiated at the mine site. The concentrates are shipped to smelters and refineries for recovery of by-product metal and production of commercial grades of refined lead.

3. Secondary Sources

Secondary lead is lead derived from salvage of worn out end-product items, such as battery plates, cable covering, pipe, and sheet, which are collected, remelted, and refined in secondary smelters to produce

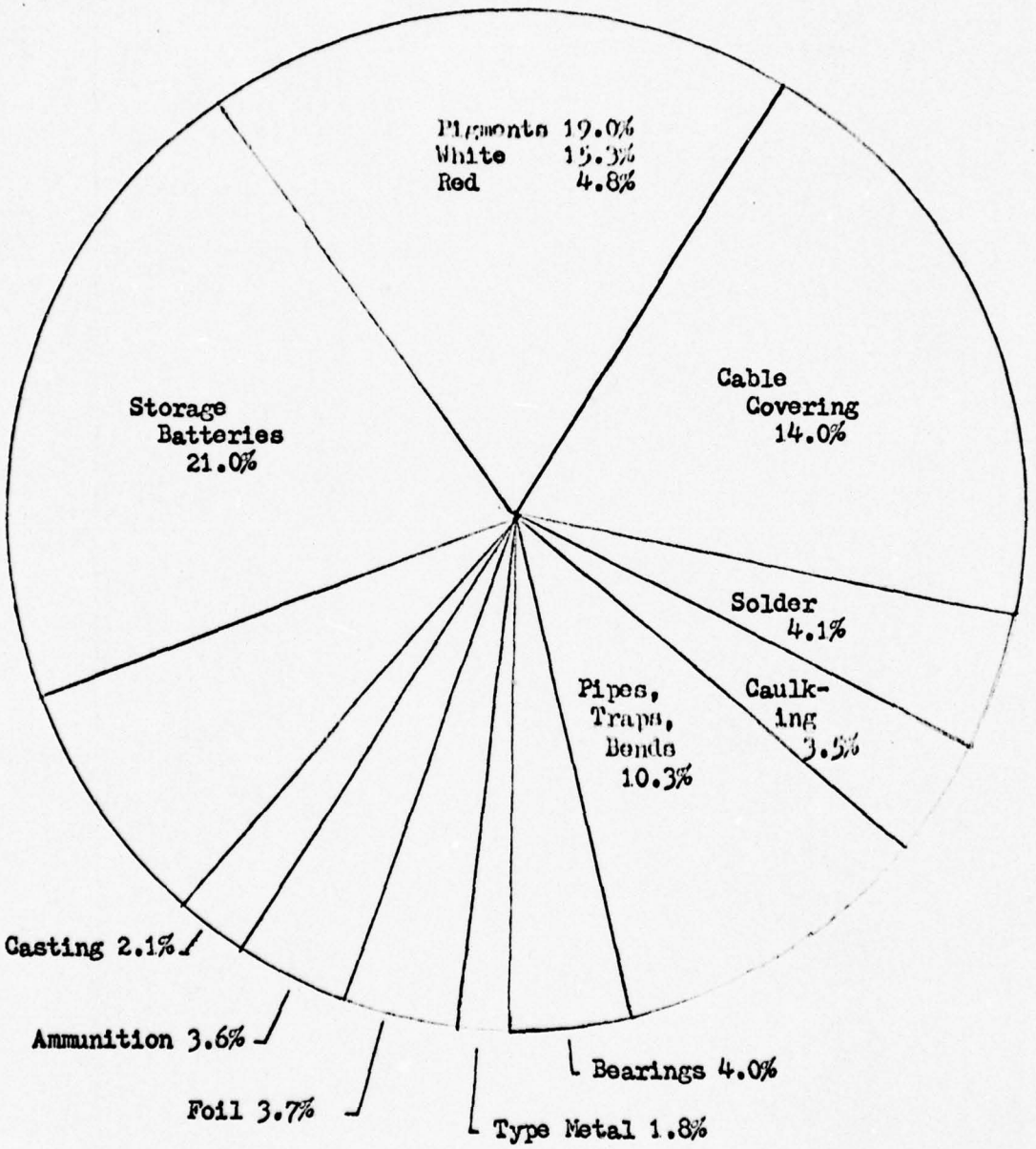
refined lead or various lead-base alloys. The secondary lead industry is of major importance in the domestic supply pattern, as lead recovered from scrap materials has exceeded domestic mine production since 1945 and domestic primary metal production since 1958. Over 200 companies process lead scrap, principally old batteries (approximately 75 percent), and produce alloy lead for industrial use. Gasoline additives and pigments are the major categories in which lead is permanently lost, and galvanizing, foil, solder, and collapsible tubes are to a large degree also unreclaimable. The National Lead Company and the American Smelting and Refining Company own and operate, through subsidiaries, secondary smelters having almost 50 percent of the secondary capacity. The remaining 50 percent is owned and operated by companies, usually limited in scope, producing various metals from secondary materials. These small operations tend to have minimal or non-existent occupational health programs and give the greatest risk of producing lead intoxication.

4. Consumption Pattern

The consumption pattern of lead has changed significantly as new technological developments have utilized the versatility of lead. The major use of lead initially was in building construction and piping, owing to its corrosion resistance and formability. Many of its present uses are recent developments. Figures 3 and 4 compare the consumption of lead by products in 1925 and 1974. Figure 5 is a composite of U. S. consumption of lead by product from 1925 to 1974. Today lead is vital to transportation, communications and electrical power transmission and is used in the medical field, atomic energy and many chemicals. Lead is

FIGURE 3: U. S. CONSUMPTION OF LEAD BY PRODUCT, 1925

Total: 857,000 Short Tons



From Minerals Yearbook (7)

FIGURE 4: U. S. CONSUMPTION OF LEAD BY PRODUCT, 1974

Total: 1,550,000 Short Tons

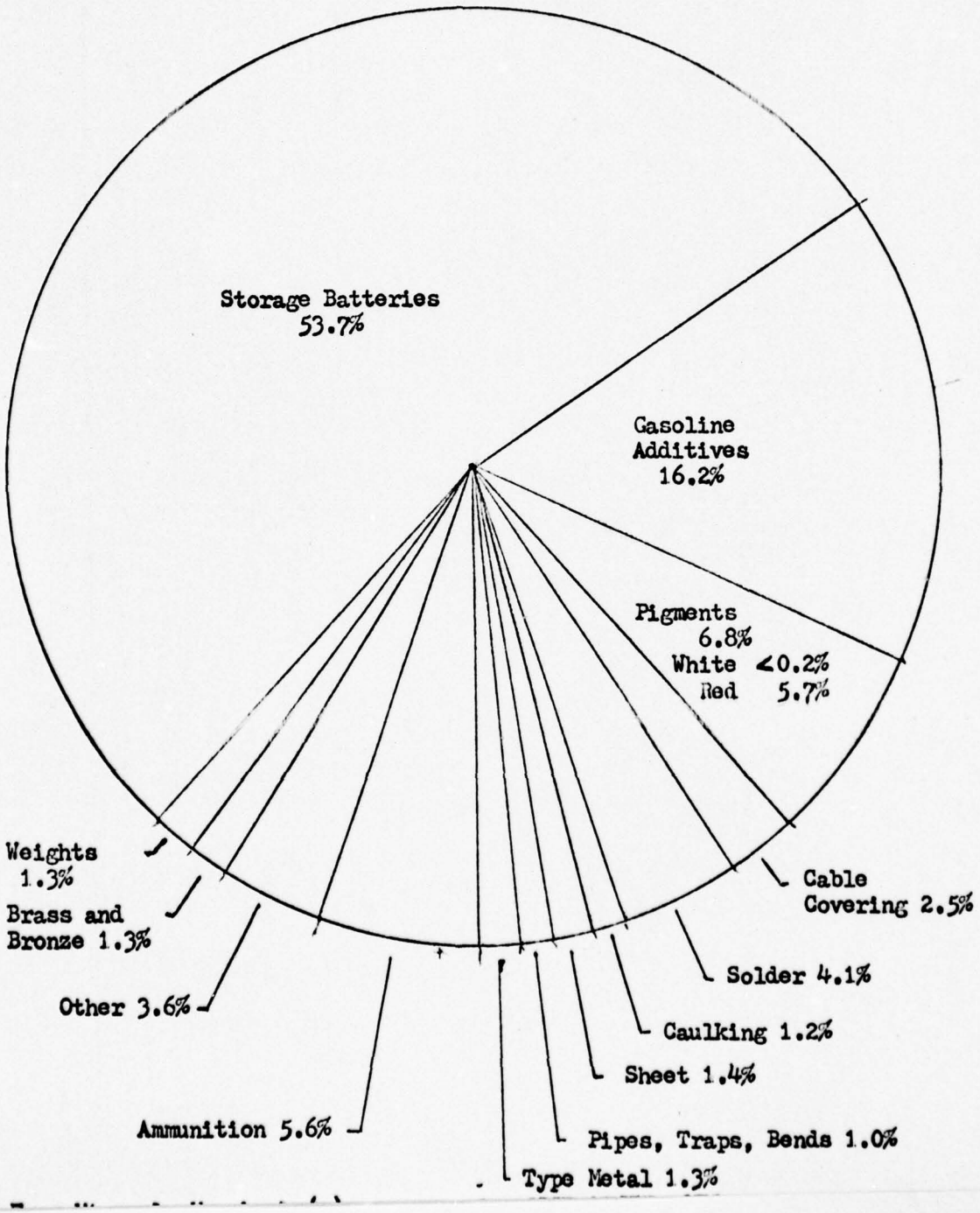
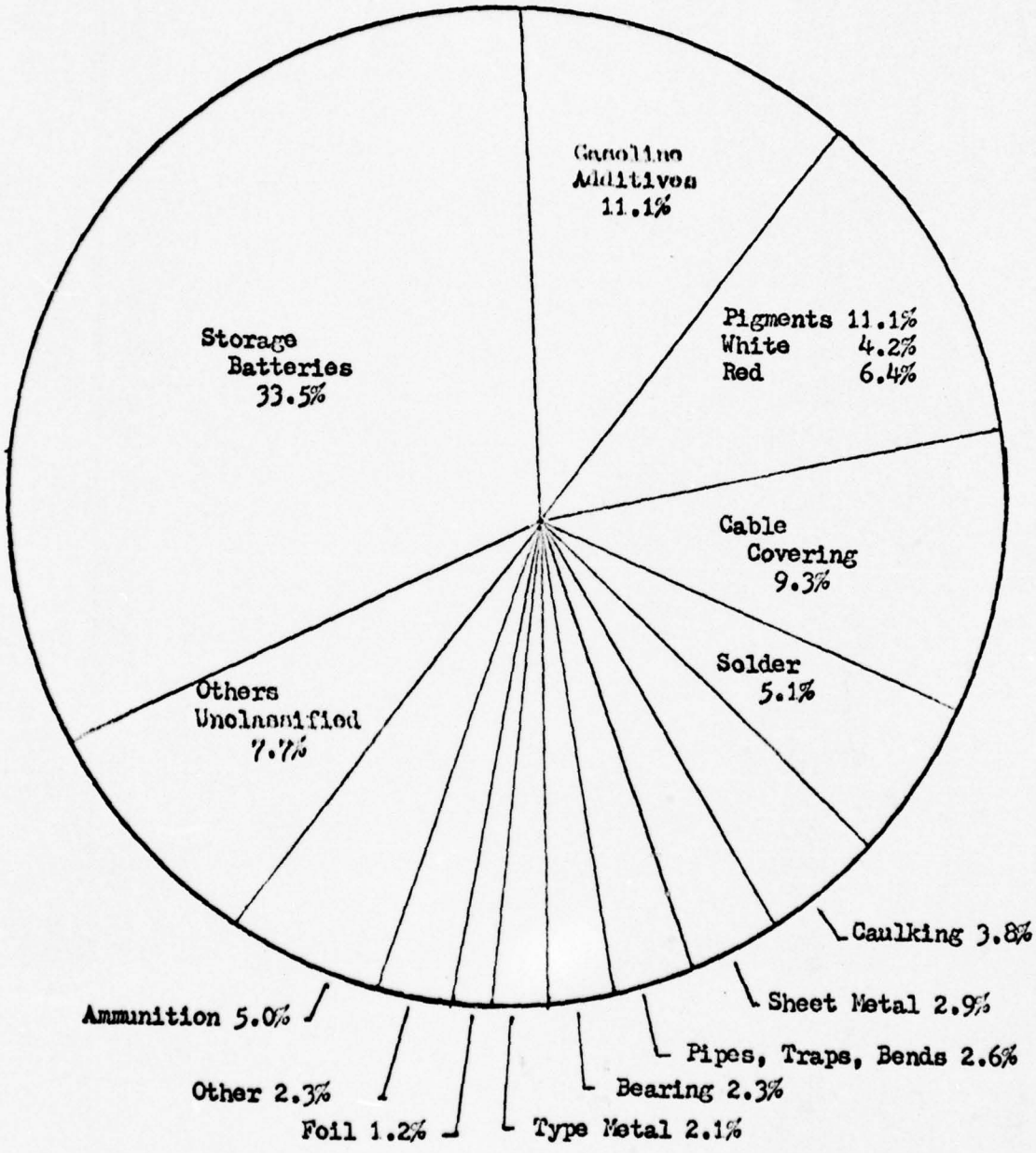


FIGURE 5: U. S. CONSUMPTION OF LEAD BY PRODUCT, 1925-74

Total: 51,518,000 Short Tons



From Minerals Yearbook (7)

still used in construction to provide noise, vibration, corrosion and moisture control. In recent years there has been an increasing demand for battery-powered vehicles for industrial delivery, material handling and sport use.

C. Lead Consumption in the United States

1. Storage Batteries

Storage batteries accounted for more than half (832,000 tons) of the total lead consumed in 1974. Over 17 million tons of lead have been used in the battery industry--or approximately 24 percent of the total lead consumption. Technological improvements and efficiency in manufacture, combined with the low-cost of materials have helped to maintain the position of the lead storage battery far ahead of its nearest competitors for portable power sources. The increasing numbers of new motor vehicles, as well as the growing number of vehicles on the road which require replacement batteries, have been the principal factors in the growth of lead storage batteries. The battery industry has continued to develop more powerful, longer-life batteries. This has been accomplished by increasing the ratio of lead oxide, the active part of the battery, over the inert part from 50:50 to 60:40. As a result, for the same amount of lead and oxide, today's automotive battery provides three times more current for starting an engine than the battery of 20 years ago. An associated market, with long range potential, is the battery-powered urban vehicle for limited range service. Such vehicles are pollution free and contribute to overall fuel economy by using off-peak power (10). Batteries are the fastest growing and largest outlet for lead throughout the world--for motor cars,

electric vehicles, and other appliances, for emergency lighting, et cetera, wherever portable power sources are required. About half of the lead used in batteries is in the form of lead alloys and the other half is in the form of oxides. The essential unit of the storage battery is the plate containing a conductive framework or grid which serves to hold the active lead material. The negative plate in the finished battery consists of metallic lead in a porous or sponge form, while the positive plate is lead peroxide. Amperage is built up by placing a number of plates in parallel. Batteries are, in fact, enclosed packages containing about 20 percent lead where little or none of the lead in them can enter the environment during their use. Virtually all the lead used in the batteries is recovered, largely within the short period of three to four years (11). The reclamation of lead is primarily from batteries and accounts for most of the recorded 23 million tons of recycled lead in the United States (7).

The problems with lead in batteries, from a health standard, arise in the manufacture of the batteries, usually a closely scrutinized and medically supervised process, and reclamation of used batteries, a process not always so closely scrutinized and supervised. Other reported cases of lead poisoning have come from the burning of battery casing for home fuel and the dicing of motor car bodies (12).

2. Petroleum

The petroleum industry used 251,000 tons of lead as a gasoline antiknock additive in 1974. Since its introduction in the early 1940's, lead as a gasoline additive has accounted for 14.1 percent of the total consumption. Comparing lead additives to gasoline with other lead products since 1925, it accounts for just a little over eight percent of the total.

Lead as an additive has slipped from a high of 280,000 tons in 1970, due to the influence of new cars burning non-leaded gasoline and a lesser number of miles being driven. The Environmental Protection Agency ruled in 1973 that lead in gasoline must be reduced by two-thirds by 1979 (13). The Clean Air Act Amendments required control of hydrocarbons, carbon monoxide and nitrogen oxides. The response of the automobile industry in meeting the 1975 standards was to employ catalytic reactors on their exhaust systems. As these are fouled by lead in gasoline, most 1975 model cars must use unleaded gasoline. The net effect of the reduced amounts of lead in gasoline is unknown. Actually, the petroleum industry uses more lead than indicated, because it employs large quantities of sheet lead and lead pipe in the construction of corrosion-resistant equipment. Lead is also used in heavy-duty greases--for example, for use under water where its lubricating properties are effective after the grease has been exhausted, and in gear compounds for drilling machinery. Approximately 2,000 tons per year are consumed in this manner, which does not apply to automotive greases (9).

Tetraethyl and tetramethyl lead have been added to gasoline because this has been the most convenient and economical method of increasing the octane ratings of all grades of gasoline which improves the performance of modern high-compression engines. Lead has good cost effectiveness in raising the octane number, is beneficial in the preparation of a balanced gasoline, provides blending flexibility in the refinery, prevents valve seat sinkage, and reduces carbon emission (14). Lead is added to gasoline in the form of tetraethyl or tetramethyl in amounts ranging from a few drops up to four cubic centimeters per gallon. Of the 5,695,000 tons of

lead that have been added to gasoline, approximately one-fourth was retained in the car--in the exhaust system, the engine oil, and filters. The rest of the lead, 4,270,000 tons, was discharged in the exhaust with the gaseous products of combustion, the lead itself being mainly in the form of fine particles of lead compounds, which constitute about one-third by weight of the solids in the exhaust. Half of the lead-containing particulates falls to the ground within a few hundred feet of roadways and is then washed away and dispersed in the soil and drains. Finer particles may be carried considerable distances before they are eventually deposited (11, 15).

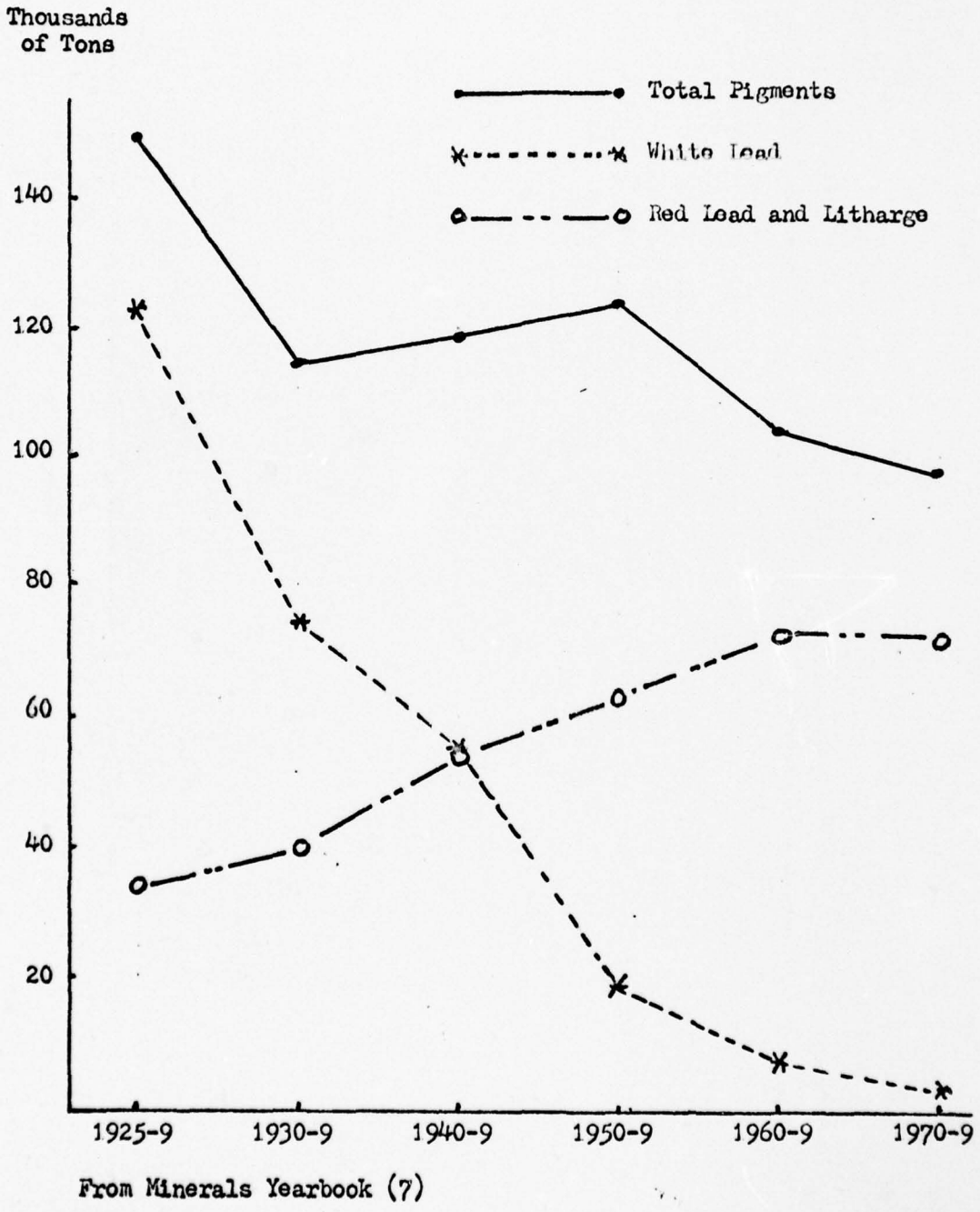
3. Pigment Colors

The paint industry, the third largest user of lead in 1974, utilized 107,000 tons of lead. It has accounted for more than 5,725,000 tons of lead since 1925 or 11 percent of the total United States' consumption. Of this portion, 2,184,000 tons were white lead. The weathering characteristics in white lead and red lead make them valuable for outdoor use. White lead, basic lead carbonate ($2PbCO_3 \cdot Pb(OH)_2$), was, in the past, extensively used as a pigment for paints. About 50 percent of the white exterior house paints on today's markets are oil based and contain amounts of white lead that vary with the manufacturer's formula. In recent years production has been declining sharply (Figure 6) and in 1974 accounted for only 2,400 tons of lead. Only part of the white lead is used in paints, the other major use being as a stabilizer in plastic sheathing for cables.

The traditional white lead paint weathered by "chalking" and so some of the lead carbonate would have been washed away by rain, to be dispersed into the environment. The widespread use of white lead paints in the past,

FIGURE 6: CONSUMPTION OF LEAD FOR PIGMENTS

Average Yearly Consumption for 10 Year Periods, 1925-1974



From Minerals Yearbook (7)

both indoors and outdoors, means there are many old buildings in which these paints can still be found, even though they may have been covered with other paints. Lead paints constitute a recognized hazard because of flaking and chalking and from the lead-containing particles released when old painted timber and painted furniture are burned.

Over 3,640,000 tons of lead have been used in red lead and litharge since 1925 (7). Red lead and the more recently developed calcium plumbate are widely used as rust-inhibiting pigments in primers for iron and steel. The primary health problem here lies with the welder who would cut through thick layers of lead-based paint or primers with an oxyacetylene flame, producing lead fumes. When welding is done in a confined space, it presents a major lead hazard and one which is difficult to control without fitting the workman with a fresh-air breathing apparatus.

Lead chromates provided some 16,000 tons (as lead) of yellow, green, and red pigment colors in 1974. These found many uses, for example, in traffic marking paints and printing inks (6). However, lead chromate is among the least soluble of all lead compounds. The paint industry uses several other lead compounds, including white basic lead sulfate and blue basic lead sulfate. The lead associated with "lead pencils" is lead chromate which has been used in the yellow paint pigment. Lead pigmented paints, like all paints, tend to be eroded and, therefore, can be said to constitute a source of lead entering the environment. Other lead pigments are used in paint for ceramics, crockery, glass, printing fabrics, artist's colors, marking pencils, engraving, and dyes. Lead is also used as a drier in paints for glazes and enamels on pottery, porcelain and chinaware.

4. Ammunition

The ammunition industry accounted for 87,000 tons of lead in 1974 and 2,600,000 tons (five percent) of the total United States' consumption since 1925. There are scores of types and sizes of bullets and shot manufactured for cartridges for the many types of guns, rifles, pistols and machine guns that exist. A number of chemical compounds of lead are also important in ammunition. Lead azide is probably most important as a detonator for explosives, while lead sulfocyanate, nitrate, peroxide and styphnate may also be employed. Antimonial lead is used for shrapnel balls and for practice bombs used by military fliers. Ammunition used to train airplane gunners is made from a mixture of lead powder and plastic so they shatter when striking the plane's special armor, instead of penetrating it. A lead poisoning potentially has been reported from lead dust in shooting galleries and there may be an additional lead body burden from eating game killed with lead shot.

5. Solder

Soldering processes used 64,000 tons of lead in 1974 and 2,643,000 tons (5.1 percent) since 1925. Lead-containing solders have many applications, mostly in industrial processes, and are used extensively in the electrical industry and plumbing and in the canning industry. The tin can industry uses 7,000 tons of lead per year just for soldering seams and closures. In addition, it uses an inseparable amount, classified under coatings, in the form of terne plate (8). For soldering, the automobile industry uses about 12,000 tons per year and in addition to the solder used for the radiators, uses lead solder to smooth out joints and other

irregularities in the body surface. Automobile body shops have, in the past, been users of body solders to help heal the wounds inflicted in modern traffic.

The most frequent source of non-industrial lead poisoning in adults is from moonshine whiskey contaminated with lead from the use of automobile radiators in the distilling process (12). In excess of 200 micrograms per liter of lead has been found in canned foods and has been attributed to leaching from the soldered seams (16). Canned milk has been analyzed to have in excess of 800 micrograms of lead per liter (17).

6. Cable Covering

Thirty-nine thousand tons of lead were used for cable coverings in 1974, and 4,780,000 tons have been used for cables since 1925 (9.2 percent). Lead is used in the construction of two general types of electrical cable--cable for communications over telephone and telegraph lines and cable for the transmission and distribution of electric power, both in overhead and underground line construction (8). There are thousands of miles of lead-sheathed cable in use today, much of it buried in the ground.

7. Construction

In 1974, 55,000 tons of lead were used in the construction industry, primarily as pipe, sheet and caulking lead. Since 1925, over 4,825,000 tons of lead have been used in construction (9.3 percent).

a. Lead Pipe

Lead pipe is used in the plumbing system to handle plumbing wastes and has been used in the water distribution system to conduct

water from the street. Supposedly, today lead pipes are seldom used in this country to carry potable water; however, many older buildings still use lead piping (18). Lead water pipes can cause problems when the water carried in them is plumbo-solvent. Considerable literature has appeared on the subject of the solvent action of certain natural waters on lead. It is generally accepted if the water is of sufficient hardness no dissolution of the metallic lead in the piping system takes place and there is no danger of lead intoxication from this source. However, water must have a total hardness of at least 50 milligrams per liter to form a protective film of basic carbonate on the insides of the pipes to prevent the lead from becoming solvent. In addition to the lead pipe used in plumbing there are lead traps, lead bends, lead vent pipes, lead floor flanges for setting water closets, lead hangers for lead pipes, and shower pans and other water-proofing throughout the system. In industrial application lead pipe is used extensively to conduct corrosive chemicals. There has been a decreased demand for lead pipe in plumbing compared to what it was in the past, but there exists a large amount of lead pipe in older construction.

b. Sheet Lead

Sheet lead is used to a limited extent in roofing, flashing, gutters and the like on the more monumental buildings, or buildings where special corrosive problems are involved. Sheet lead is used principally in the various chemical industries handling sulfuric acid, as well as other acids, plating baths, and tanks for chemical processing. Lead is also used in vibration and radiation isolation. The widest use of lead in isolating vibration is for the foundations of buildings adjacent to

railroad tracks or other sources of vibration. Similarly, lead is frequently used under machinery foundations in buildings to prevent the vibration of machines from being transmitted to the building.

c. Caulking Lead

Caulking lead (9,982,000 tons since 1925) is simply soft metallic lead whose main use is to make joints in cast iron water mains, sewer and soil pipe.

8. Type Metal

Type metal accounted for 20,000 tons of lead in 1974 and over 1,100,000 tons (2.1 percent) since 1925. A modern newspaper plant may use 100 tons of stereotype metal cast several times over within a 24 hour period. In an *in vitro* study of the extractability of lead in printed matter it was found that up to 200 micrograms of lead could be extracted from "small" pieces of paper at pH values in the range of human gastric fluid (19).

9. Brass and Bronze

Twenty thousand tons of lead were used in brass and bronze in 1974, either for the properties it imparts to bronze for bearings or for free machinability in brass. Statistics available only since 1951 show that 534,000 tons have been used in conjunction with brass and bronze.

10. Weights and Ballasts

Twenty thousand tons of lead were used in weights and ballasts in 1974. Statistics available only since 1951 show that over 300,000 tons of lead have been used as weights and ballasts. The use of lead as weights

or balances are numerous; a few are balances for: machinery, locomotive and train wheels, automobile wheels, anti-aircraft guns, diver's equipment, industrial trucks, airplane control surfaces and propellers, naval ballasts, fishing sinkers and seine weights.

11. Bearings

Bearings accounted for 14,000 tons of lead used in 1974 and 1,209,000 tons (2.3 percent) since 1925. Lead-base alloys are being used today in almost all kinds of bearing applications. Among these are automotive connecting rod, main and camshaft bearings, diesel engine bearings, steel mill bearings, older railroad car journal bearings, all kinds of electric motor bearings and a variety of other industrial applications.

12. Miscellaneous

Of the total 1,550,000 tons of lead used in the United States in 1974, 40,000 tons have not been accounted for and will be lumped together as miscellaneous and only the more important uses will be discussed in any detail.

Approximately 2,500 tons of lead have been used in pesticides per year (decreasing since 1946). Some of the compounds used as pesticides are lead arsenates, lead phenolates, lead nitrates and lead monoxide.

A growing use of lead is as a stabilizer in plastics. While the lead content is usually small, amounting to only a few percent, the total use for this purpose amounts to several thousand tons per year. The principal use is in vinyl plastics for electrical cable insulation, but it is also used in plastics for many other purposes such as floor coverings, hose pipe and sheet.

A number of commercial uses exist for powdered lead. Lead powders

are used as a constituent of bearings, brake and clutch facings, as a plastic filler, in the manufacture of rubber, free machining steel, paints, lubricants and pipe joint compounds.

Lead wool is used for the same purposes as caulking lead, but since it is used in solid form without heat it can be employed under water or in gaseous locations where heat would be dangerous or impossible to use. It is also used for plugging oil wells. Lead is used for molding plastics, rubber hose, and in impression lead used in the printing trades, as well as dyes for stamping aluminum.

Lead foil, collapsible tubes and seals all play important roles in modern packaging. Lead collapsible tubes are used for paste products, such as mucilage, shaving and dental cream and colors-in-oil. Lead foil is used by the Military Ordinance Department for packaging various ordinance materials. It is used for packaging x-ray and Polaroid film. In addion to packaging, foil is used extensively in the manufacture of paper-wound condensers and mica condensers for use in various electronic circuits including television and radio sets. Lead foil is used in metallic packing. Also, it was a familiar sight at Christmas time as a Christmas tree decoration in the form of tinsel (20).

The use of lead in the ceramic and glass industry has increased substantially in the last few years and now consumes upwards of 30,000 tons per year, mostly in the form of oxides and silicate, many which are imported and do not show in the consumption statistics. The finest glass tableware and optical glass, as well as most glass for electrical and shielding purposes, may contain large amounts of lead. Glazes for china and many structural clay products may also contain lead. Lead is also used in electronic applications, such as lead zirconate-titanate, as piezo-electric

product in ultrasonic cleaners and lead telluride in thermo-electric materials. Many other chemical compounds of lead are of considerable commercial importance. Lead is used in bronzing gold pencils, mosaics, printing and photography. It is used in the manufacture of matches, explosives and fireworks. Also, lead, in varying amounts, is used for casting metals, manufacturing chemicals, annealing, galvanizing, and lead plating.

D. Industrial Lead Poisoning

In 1913 at least 1,770 cases of lead poisoning in the U.S.A. were reported in 19 plants employing some 7,500 persons. These included 41 encephalopathies, 35 cases of paralysis and 16 deaths (21). These figures contrast greatly with the present situation where cases of industrial lead intoxication rarely occur in the over 1.6 million workers exposed to lead (22). The recently reported cases of industrial lead poisoning in no way correlate to the severity of cases reported 30 to 40 years ago. The alleviation of industrial lead poisonings has been mainly due to the advances in occupational medicine, which have influenced regulations and compensation for injury and the installation of various protective measures, which have greatly reduced the incidence of toxic manifestations.

In the past, industrial lead poisoning has chiefly been caused by the presence of finely divided lead compounds in the air. One of the greatest dangers has been in smelters from the fumes of lead oxides and sulfates from the furnaces, and next to that in the fine dust from the flues and baghouse. Lead burners, who melt together two surfaces of lead with a high temperature torch, might be presumed to have some of the most

dangerous work among the metallic lead occupations if there are high temperatures involved. Another potential hazard would be tempering metallic objects in a bath of lead, and later on brushing off the gray oxide coating which has formed. The very lack of evident and immediate danger in the use of metallic lead seems to induce a neglect of ordinary precautions (21). Today the main problem in industrial lead poisoning is presumed to be with the small plants which lack a comprehensive occupational medicine program.

E. Summary

Lead is ubiquitous to our everyday life, perhaps far more than we realize. We have long associated lead with industrial processes; but it, and its alloys, are all around us. Unfortunately, it is not always easy to recognize. About a third of its use is in chemical compounds that bear no resemblance to the metal itself. Often it is in alloys with other metals or is only a hidden component of some device we commonly employ.

Since 1760, when the lead industry began in this country, 94 billion pounds (this figure excludes all lead that has been reclaimed) of lead have been consumed in products that are now in our environment. Some of it remains in our structures; but because lead does not deteriorate or decay, much of it lies where it was discarded in our soil and rubbish heaps. It is necessary when evaluating lead body burdens to consider environmental contamination as well as industrial processes. Sources of lead are all around us.

III. LEAD IN THE ENVIRONMENT

A. Introduction

Lead occurs everywhere in the environment--in the soil, in the water, and in the atmosphere. It has been detected in the remotest regions of the world and is found both as a natural constituent and a contaminant. Lead is found as a natural constituent of the atmosphere at 0.0005 micrograms per cubic meter of air (23), with its primary sources being airborne dust containing 10-15 parts per million lead (24) and gaseous diffusion of lead from the Earth's crust (25). Lead has occurred in the atmosphere throughout geologic time. By analysis of annual ice rings in the interior of Northern Greenland, it has been found that lead concentrations in the atmosphere have increased from less than 0.0005 micrograms per kilogram of ice at 800 B.C. to more than 0.2 micrograms per kilogram in 1965 A.D. At the beginning of the Industrial Revolution, in 1750, the ice layer showed a lead concentration 25 times greater than the natural layer (9).

Lead has long been recognized as an industrial poison, but it has not been until recent years that the effects of lead as an environmental poison have been considered. Man's activities have tended to increase the lead levels around and within his domicile. Industrial production of lead and its products have caused contamination of the atmosphere with subsequent fallout to the surrounding soil and water.

The metabolism of lead in man has been studied intensively. Lead which travels undisturbed through the intestines or falls upon the skin to be washed off has no practical health concern. It is only when lead is absorbed into the internal environment of the body that one begins

to think in terms of a lead body burden. Quantities of lead ingested or inhaled from the general environment are thought to be small, but merit consideration because of the total and cumulative effects of lead in the body.

B. Soil

Lead occurs naturally in the soils of the Earth with an average order of magnitude of 16 parts per million (26). There appears to be a natural mechanism moving lead upward in the crust of the Earth (9). In virgin soils Kehoe found a range of 0.07 to 30.0 parts per million (27). Maximum values for natural soils have been reported over 200 parts per million (28, 29). Soils near natural deposits of lead containing ores have been reported up to 10,000 parts per million (30). No soil samples of sufficient size have been found to be free of lead.

A range of lead concentrations have been found in contaminated soils by many investigators. Soil contamination by lead can be divided into two categories: burned leaded fuel and industrial processes and products. The burning of leaded fuel emits lead into the atmosphere primarily from automobiles and bears a direct relation to the density of the traffic and inverse relationship with distance from the traffic. The surface soil of parks, narrow bands along major thoroughfares and street dust in larger cities are heavily contaminated (9, 18, 31, 32, 33). Of the two categories, the lead-using industries have contributed two to three times as much lead to the soil environment as have the leaded fuels (9), mainly in the form of paint pigments and metallic products. The highest reported concentration of lead from contaminated soil has been due to the contribution from the natural weathering of house paints (18, 34). The highest

reported concentrations of lead in soil due to contamination have been 20,000 parts per million (34). Because of the insolubility of the lead pigment, it may remain in the soil long after the paint source has been removed. The concentration of lead in the soil from weathering house paint is dependent upon the lead content of the paint, the distance from the house, the size of the house, the degree of weathering, the maintenance level, and the meteorological conditions. Smelters and lead processing operations contribute to soils downwind from them. Urban soil containing high concentrations is a potential major environmental lead source for children (18).

C. Air

The concentrations of lead in ambient air in U.S. cities range from one to five micrograms per cubic meter (18, 32, 35, 36). High concentrations of 8.7 micrograms per cubic meter from London (37) and 8.9 micrograms per cubic meter from Tokyo (38) have been reported. Nearly 98 percent of the airborne lead that can be traced to its source comes from the combustion of leaded motor fuel (9). Air over heavily traveled streets contains more lead than that above lightly traveled ones; also, air in urban areas contains higher concentrations of lead than that in rural areas (32). About half of the lead-containing particulate matter from automobile exhausts is removed from the air by gravity within a few hundred feet. The air over the largest American cities has a concentration of lead 20 times greater than the air over some sparsely populated areas of the country and nearly 2,000 times greater than the air over the mid-Pacific Ocean (9).

Lead compounds in the atmosphere are dispersed from many other minor sources: dusts produced by the industrial processing of leaded products

usually contained in the near or immediate vicinity of the processing plant (39); by the exhausting of workroom atmospheres; the weathering of lead-containing products with the resulting flaking and distribution into the atmosphere of a portion of the lead-bearing dusts, such as paint, solder, etc.; the abrasive action of automotive traffic on lead-painted lane dividers on streets and highways; the combustion of coal and suspended soil particles high in lead concentrations; the incineration of leaded plastics and other materials whose usefulness has ended; the burning or sandblasting of lead-painted surfaces of houses, bridges and other structures before repainting; the recovery of lead from old battery cases, lead pipe, lead-sheathed cable, and sheet lead in secondary smelters; and, on a small scale, the welding and soldering operations conducted in plumbing and electrical repair shops (9, 32). The magnitude of these additional sources of emission could be considerable, but it is unknown.

There is somewhat of a controversy about the role that atmospheric lead plays in environmental lead poisonings. One group contends that under ordinary circumstances the amount of lead that comes into effective contact with the body through respiration is only a small fraction of the total lead taken in through all sources (14, 40, 41, 42, 43, 44, 45). Their contention is that there is no conclusive proof that the current atmospheric lead endangers the health of either adults or children. The other group argues that there is a significant number of low-level lead intoxications in children which cannot be accounted for by pica alone. The atmospheric concentration contributes to the total lead body burden and may be a significant factor in environmental lead poisoning (46, 47, 48, 49, 50,

51, 52, 53, 54, 55, 56). They further argue that because leaded gasoline is the primary source of atmospheric lead and because lead is not indispensable to gasoline, its use in this application should be discontinued.

Upon reflection of the dissenting scientific opinions, one is reminded of the debate that occurred in the early 1920's concerning whether or not to restrict or prohibit white lead paint. At that time the concern was for the painter and the arguments went:

> "Any prohibition of the use of white lead in the painting industry would practically mean a calamity for the consumers of paint, as the result would be less permanent and satisfactory work, for the reason that white lead, when used either alone or as a constituent of prepared paints, meets requirements that in the present state of the art cannot be practically met by the substitution in substantial amounts by other pigments for it.... The risk of lead poisoning can be entirely removed if the workers will observe scrupulous personal cleanliness. If this were done any cases which did arise would be due solely to the worker's own carelessness... to describe painting as a dangerous trade is merely an arbitrary statement.... White lead could no longer be regarded as dangerous to health, thanks to the improvements in its manufacture... dangers exaggerated... infringement of liberties...." (57).

In retrospect, it is easy to see the fallacies of the above arguments. If the truth had been realized then, many cases of pediatric lead poisoning would never have occurred.

It is clear that one major source of general environmental contamination is the combustion and dispersion of lead alkyl compounds used as automotive fuel additives. Much speculation has been offered as to their contribution to the total daily assimilation of man; however, factual data is very limited and mostly unknown. In children, also, the extent to which atmospheric lead in congested urban areas contributes to increased

lead absorption and lead poisoning is not clearly defined; here, however, many authorities feel that atmospheric lead due to leaded gasolines could be significant.

D. Water

The lead content of natural waters ranges from 0.001 to 0.06 milligrams of lead per liter of water (1, 27, 58). The natural or treated water supplies available for use in the United States usually contain about 0.01 to 0.03 milligrams per liter (59), well below the United States Public Health Service's standard for drinking water of 0.05 milligrams per liter. Consequently, water does not constitute a significant source of lead (1). Lead in surface waters is usually associated with sediments of suspended solids rather than dissolved in water. Because most of the lead entering the aquatic system via precipitation and runoff is not water-soluble, it is apparently removed from the water by sedimentation (9). Common treatment processes used by water suppliers seem to be very effective in removing lead (60).

Lead contamination in drinking water has been detected due to soluble lead salts in the luting compounds used in the joints of water pipes, lead piping, especially new lead pipes, and leaded vessels used to store or carry water. If the water is allowed to remain stagnant in these systems for prolonged periods of time, excessive contamination by lead can occur. There have been reported cases of lead poisoning among the residents of new housing developments in which inadequate care was exercised in the general water distribution system. Similar conditions exist where extensive lead piping has been used (61). Up to 100 micrograms of lead per liter of water have been found in lead-lined storage tanks and lead piping (62).

Water can also extract lead from leaded plastic (viniplast) pipes and containers when in contact for extended periods of time; therefore, plastics containing lead are not recommended for use in water storage and piping (63, 64).

E. Food and Drink

Kehoe, et. al. (27) found lead in every food item that was obtained from the fields and dwellings of the inhabitants far removed from society and any commercial source of lead. Kehoe (1) has reported that the lead content of the composite food of adults in the United States ranges from less than 0.1 milligram to more than 0.4 milligrams per day with an average of 0.3 milligrams per day. Lead is found to occur in all foods, whether of vegetable or animal origin (1, 58, 65, 66, 67, 68, 69, 70, 71). Lead in food can result from the biologic uptake from soils into plants consumed by food animals or man, usage of lead arsenate pesticides, inadvertent addition during food processing and by leaching from lead soldered containers or improperly glazed pottery used as food storage or dining utensils.

1. Lead in Plants

Lead in plants can be attributed to the uptake from the soil either naturally or contaminated from spraying or industrial processes, or from water, either rain water or ground water. Lead on plants is due primarily to fallout from atmospheric lead. Each source of lead may in turn affect the roots, stems and leaves of plants in a different manner. The low solubility of lead in water affects the ability of plants to assimilate lead. The primary source of lead uptake in plants is thought to be due to rainfall (72). Widely varied concentrations of lead in soil

have little effect on the lead content of plants. The naturally occurring lead in soils is mostly unavailable to plants as is the atmospheric lead along heavily traveled highways. Leafy portions of plants within a narrow band of busy highways contain larger concentrations of lead than do plants growing farther away--most of which can be washed away with water (9). The amount of lead contamination on fruits and vegetables is a function of the distance from the highway, the extent of the surface area exposed, the nature of the collective surface, duration of exposure, motor vehicular traffic density, and meteorological conditions (73, 74).

2. Lead in Animals

Lead does not tend to accumulate in the flesh of animals. Lead is a bone seeker, and as a general rule, the accumulation of lead in the bones of vertebrates as well as fish exceeds that in muscle by a factor of 50 (9).

3. Lead from Containers

High concentrations of lead have been found in commercially canned foods due to the leaching of lead from the soldered seams of the walls of the cans. Storage after opening the can of produce without pouring the contents greatly increases the dissolution of lead from the seams and the walls (75). Of particular concern has been the finding of high concentrations of lead in baby food (72, 76, 77, 78, 79, 80), primarily fruit juices and milk. A canned tomato product was analyzed and was found to contain over 5,000 micrograms of lead per liter (16). Dangerous levels of lead in toothpastes have been reported in other studies (81, 82). Acidic foods and beverages, including tomatoes, tomato products, sauerkraut, most fruits

and fruit juices, cola drinks, coffee, such alcoholic beverages as wine and cider, and pickles and relishes using vinegar, can dissolve the lead in improperly lead-glazed earthenware containers (83, 84, 85, 86, 87, 88, 89). Also, it has been reported that the ingestion over a period of time of acidic foods cooked or stored in leaded pewter pots could lead to poisoning (90). On a recent request from the Army Air Force Exchange Service, the Dallas District Office of the Food and Drug Administration analyzed silver-plated cups and goblets sold world-wide in Post Exchanges and found excessive levels of leachable lead in the articles examined. Some of the apparent lead sources were the base metals, spoils and fluxes used to manufacture this holloware (91).

4. Illegal Whiskey

The magnitude of the lead intoxication problem in moonshine whiskey drinkers is unknown, but it is suspected that it may be significant, especially in the Southeastern part of the United States. Lead is a common contaminant of the illicitly distilled whiskey manufactured in the United States. The contaminant comes from the method of construction of the "stills." Frequently, old discarded automobile radiators are used as condensers, while the other components are generally connected together with lead soldering. The metallic lead is attacked slowly, but surely, by the hot vapors and condensing liquor. Although the forms in which lead is present in liquor have not been identified, it is thought that acetic acid in the distillate may react with lead to form lead acetate (92). Regardless, the concentrations of lead in moonshine whiskeys have been analyzed to show up to five milligrams per liter of sample (93).

5. Pica

The most serious and crippling current form of lead poisoning is encountered in infants and young children living in deteriorating houses in the cities. This source of poisoning is known as pica. "Pica" has been defined as the repetitive ingestion of things that are not food (e.g., dirt, cigarette butts, plaster, paper, putty, paint chips, wood, and clay). Today in the United States, lead poisoning in children is believed to be due almost entirely to the repetitive eating of leaded house paint. The clear association between lead poisoning in children with pica and old deteriorating and dilapidated urban housing is well-documented (18, 94, 95, 96, 97). Lead in paint chips, plaster, caulking and dirt ingested by children with pica is the prime causal factor in childhood lead poisoning, with the lead in paint being the usual indicated agent (98). Additional lead may have been ingested by people who chewed pencils covered with leaded paint (99, 100, 101).

Federal regulations have been established to limit the amount of lead which may be added to paints that are intended for residential use to 0.06 percent lead. The intent of such limitations is to curtail the incidence of present and future lead-based paint poisonings of children. Before 1940, lead pigments were widely used in both interior and exterior paints. As recently as 1958 lead-pigmented paint could be bought for use as an interior paint (102). Today, the bulk of lead-based paints are those used for specialty coating--rust preventives, moisture resistant paints, decorative gloss paints with exotic tints, etc. However, a study by the U. S. National Bureau of Standards reported that violations of the statutory lead-warning label requirement were widespread (103). Generally,

though, the source of lead in lead-pigmented paints is considered to be the paint applied to woodwork and plastered and papered walls of dwellings prior to World War II. In such paint, lead may constitute five to 40 percent of the final dried solids. Hazardous amounds of lead paint are still available in many old houses and are readily accessible to children because of deteriorating conditions (104).

In 1974, a report of the Ad Hoc Committee to Evaluate the Hazard of Lead in Paint prepared by the National Academy of Science for the Consumer Product Safety Commission recommended reducing the permissible lead content of paints from the present 0.5 percent to the "lowest practicable" concentration. The report stated that inadequate scientific data on lead poisoning prevents setting a more precise, scientific standard at this time. Special emphasis should be given to learning the lead absorption rate of children--the primary victims of lead poisoning (105).

The hazard presented by lead-based paint is related both to the lead content of the paint and to poor maintenance of painted surfaces. It is easy for a child to pick and eat loose flakes from chipped and deteriorating painted surfaces. The lead content of such paint flakes is far in excess of the amount likely to be inhaled, even in congested areas. Therefore, it seems clear that the principal cause of clinical cases of lead poisoning in children is the direct ingestion of lead-containing paint chips.

6. Cosmetics

Some commercial hair sprays contain large amounts of lead. The solder of the cans gradually dissolve and increase the lead concentration

of the contents. The rate of dissolution is related to the resin constituent of the hair spray (106).

F. Summary

The role of naturally occurring lead in the environment is not considered to be a major factor in lead poisoning. The contamination of the environment with lead by industrial processes and the combustion of leaded gasoline plays a more significant role, but their exact contribution to the total body lead burden is unknown. The major concern is the lead contained in products applied to the home environment and the contamination of foods and beverages from leaded containers used in the manufacture, storage and processing of products for consumption. Alcoholic beverages and acidic foods and drinks are especially apt to dissolve lead from the containers. Practically all wares made for food and beverage service, which have been involved in incidents of lead poisoning, come from small studio potteries, hobby shops, or homemade ware, classrooms, foreign folk or specialty shops or from imports. Untrained people should not make glazed ceramic articles intended for food or beverage use. Infants and young children are believed to be at an increased risk to lead poisoning because of pica.

IV. LEAD POISONING

A. Introduction

1. General

Lead poisonings have plagued mankind throughout recorded history. From antiquity there has been knowledge that lead compounds are toxic. It is now known that lead in all forms can be poisonous. Lead is especially hazardous because the poison is cumulative, the toxic effects are insidious and are apt to go unrecognized and because the very young tend to be especially susceptible. Another inherent danger in lead toxicity is that the population is not always aware of its exposure. Lead and its products take many forms, many of which are not easily recognized. Lead poisoning is caused by processes and contamination produced by man, and, therefore, should be preventable.

2. Background

Lead, like many of the elements man has adapted for his use, may be harmful as well as beneficial. Knowledge and clinical studies of lead poisoning date from the 17th Century, when, in addition to industrial plumbism, outbreaks of lead poisoning occurred throughout Western Europe as a result of the addition of lead to wine to retard fermentation and because of its employment in the manufacture and storage of cider and in material for cooking vessels and other household articles (3). The advent of the industrial revolution brought an ever increasing demand for the heavy metals, lead included. The steadily increasing employment of lead for household and industrial purposes led to an enormous increase in the number of lead poisonings. In the United States in 1913, Alice

Hamilton reported there occurred at least 1,770 cases of lead poisonings in 19 plants employing some 7,500 men; among these were 41 encephalopathics, 35 cases of paralysis and 16 deaths (21).

Occupational medicine has made great strides in the lead industry in this country. No cases of death attributed to industrial lead intoxication have been reported in the recent literature. Indeed, one must scrutinize the literature carefully to find reports of lead poisoning in this country. Cases that are reported in the current literature are generally mild and in no way approach the severity of cases reported just a few years ago. In fact, most cases that are reported show that occupational absorption of lead is some degree of elevation above that of the normal individual in the general population. Perhaps, the exception to this is pediatric cases of lead poisoning attributed to pica.

A considerable share of the responsibility for the dramatic decrease in industrial lead cases must go to scientists involved in industrial medicine, toxicology, and hygiene who have persisted for the last 50 to 60 years through research, investigation and consultation on regulation. Through their efforts, there are now industries using large quantities of lead and its compounds in potentially dangerous operations where no case of even the mildest type of lead poisoning has occurred in many years. This has been accomplished by innovative process changes and engineering controls whereby exposure to the workers has been minimized. Operations that require exposure are monitored by medical surveillance programs to insure workers are afforded maximum protection. Though medical surveillance is important, the principal cause is reduction of exposure through process change and engineering control. The industrial hygiene

surveillance monitors the controls, and medical surveillance monitors the entire program. Usually, though not always, the lead intoxications seem to come from smaller industrial plants where the occupational health program is inadequate or non-existent.

B. Lead Metabolism

1. Route of Entry

Lead compounds enter the body most commonly through the gastrointestinal tract and/or the respiratory tract (1). Kehoe has estimated the "normal" adult daily intake of lead from food and beverages and in the air to be on the order of 330 micrograms per day. Food and water are generally considered greater potential sources of lead in man than air; yet, there are many uncertainties as to their relative contributions, mainly because of the imperfect state of knowledge concerning the fate of the lead that is inhaled (9). The major route of entry for lead in the industrial worker is the lungs. Information from the study of industrial populations commonly relates the clinical or biochemical findings to the concentration of lead in the air. Similar relations between the amount of lead absorbed and the biologic effect produced have been found for ingested lead (1).

a. Respiratory Tract

Precise knowledge concerning the contribution of inspired air to the total lead body burden is inadequate to allow anything more than general approximations (9). Actual deposition of lead in the respiratory tract has been shown to be approximately 37 percent (1, 107), varying with particle size. The degree of absorption is dependent upon the

proportion of the dust particles that are respirable (less than five microns) as well as the respiratory volume. Little is known of the fate of the lead once it has been deposited within the respiratory tract. It has been estimated that 40 percent of the lead deposited in the airways is transferred to the gastrointestinal tract.

Clinically evident disease due to inhalation of inorganic lead salts does not usually occur unless the concentration in air exceeds 0.5 milligram per cubic meter--roughly 100 times the maximal concentration reported in the ambient air in urban areas and 1,000 times that reported in rural ambient air (9).

b. Gastrointestinal Tract

Food, water and other beverages are the major sources of lead input in man and occur via the gastrointestinal tract. The best source for information on the absorption and excretion of lead in man comes from Kehoe's balance studies conducted at the University of Cincinnati's Kettering Laboratory (1). The results of these studies over a span of many years show that at a normal intake the amount of lead excreted generally exceeds slightly the amount ingested in food, water and incidental beverages. The slight greater difference in output over input has been thought to be attributable to lead inhaled in air. Kehoe's data infers that net absorption of lead from the gastrointestinal tract is somewhat less than 10 percent, some of which is excreted back into the alimentary tract and evacuated. The absorption of lead from the gastrointestinal tract appears to be regulated by the mechanism controlling calcium and phosphorous absorption. Approximately 90 percent of the

ingested lead is eliminated in the feces with about 10 percent voided in the urine. There are other slight losses, of which sweat is the most perceptible. The concentration of lead in sweat is much the same as that of the urine. Lead is lost in falling hair, and discarded and desquamated skin to some variable, imprecisely known degree.

a. Skin

Absorption of lead through the skin and other possible portals of entry has been a matter of much speculation and investigation. It appears, though, that penetration through the skin is a factor only when organic lead compounds are involved (1).

2. Metabolic Effect

Lead absorbed by the pulmonary blood stream is transported to various organs and tissues. More than 90 percent of the lead in the blood is held by erythrocytes, but lead has a preference for the bone and thus will accumulate in the bone tissue (108). Over 90 percent of the human body burden is found in bone. In descending order, the lead concentration was highest in the aorta, liver, kidney, pancreas, lungs, bone, spleen, testes, heart, and brain in samples taken from 150 cases in nine American cities (12). Increasing concentrations of lead have an adverse metabolic and functional effect of inhibiting the formation of heme which is largely responsible for the anemia of lead poisoning. Classical lead poisoning in both human and experimental animals is characterized by the accumulation of non heme iron, protoporphyrin IX, and delta-aminolevulinic acid in red blood cells (109) which leads to a shorter life span of erythocytes and impairment of hemoglobin synthesis. Serum iron may be increased in humans

with lead poisoning, but without iron deficiency states. These hematopoietic effects occur early before the typical signs and symptoms of lead intoxication are present, and are, therefore, important for diagnostic parameters (110).

C. Types of Lead Poisoning (Signs and Symptoms)

Industrially induced lead intoxication is generally well defined and the laboratory results are usually conclusive (111). However, low-level environmentally produced lead intoxication can display a wide variety of symptoms. Childhood lead poisoning is a type of plumbism which is especially treacherous and in which the sequelae and fatality rates remain disturbingly high (1).

1. Alimentary Tract

The most common type of lead poisoning is expressed in alimentary tract symptoms. The chief early symptom is a loss in well-being. The first symptom is usually loss of appetite, especially upon first arisal. Insomnia with bodily discomfort and cramps in the leg muscles often occurs. Constipation is usually present (even though diarrhea has been found to occur) with colicky abdominal pains which may present with sharp onset and recurrent spasms with the patient in excruciating pain. The patient may writhe in pain, retract his legs spasmodically into his abdomen, groan, clinch his hands, and grit his teeth, while beads of sweat cover his brow. This distress may subside and recur or it may be almost continuous (1, 112, 113, 114, 115, 116). The general non-specific symptoms are usually considered as signs of poisoning (117). If history reveals an exposure to lead and laboratory analysis shows increased lead

levels in body fluids, the diagnosis would tend to point to increased lead absorption. These symptoms are reversible and complete recovery is possible once the patient is away from the lead source.

2. Neuromuscular

In the neuromuscular type of lead poisoning the neuromuscular symptoms overshadow the alimentary symptoms. Greatly increased muscular tone with spontaneous pain in muscles and at joints and greatly increased reflexes in the extremities may occur; however, most commonly there is a general weakness, lowered tonus and atrophy of extension muscles which may develop into "wrist drop" or "foot drop." This type of disability is usually irreversible. Perhaps one of the most significant diagnostic symptoms is that there are usually complaints of generally severe fatigue and of weakness which seems disproportionate to the severity of the illness (1, 118).

3. Encephalopathy

Encephalopathy (119), the third type of lead poisoning, chiefly occurs in children and is manifested by headache and insomnia; persistent vomiting, which may be projectile (typical lead colic may or may not be present); visual disturbances with choked optic disks; irritability, restlessness, delerium, hallucinations, convulsions and coma; intracranial pressure is characteristically high with the cerebrospinal fluid generally unremarkable except for an elevation of total protein. Death occurs from exhaustion and respiratory failure (241). The mortality rate is high; recovery is slow and frequently incomplete. Mental enfeeblement is a common sequel; this occurs in 25 percent of the cases (9).

4. Present-Day Plumbism

Kehoe (120) further delineates a current type of lead poisoning. There are numerous variations in the expression of lead intoxication, but it is usually one of a systemic poisoning. In addition to the above mentioned types there may be quite pronounced hypotension associated with bradycardia indicative of vascular involvement. There also seems to be a localized vaso-constriction which causes facial pallor (saturnine facies) and pallor of the eye grounds with retinal hemorrhages.

5. Pediatric

Infants and young children (less than four years) appear especially susceptible to the toxic effects of lead. Lead poisoning in small children can be so traumatic as to cause mental enfeeblement and even death. The early symptoms of lead poisoning are subtle, subjective and non-specific and consequently are more difficult to recognize in children. There are many reasons why young children may be more susceptible to lead. Among these are: greater vulnerability of young growing tissue, greater variation in gastrointestinal acidity or alkalinity to include pH ranges likely to dissolve lead, shifts of lead into and out of growing bones, and inherent sensitivity of a child's nervous system to lead (121). Therefore, a review of the manifestations of pediatric plumbism and its difference from the adult disease is given. Gastrointestinal symptoms are recurrent--vomiting, vague abdominal pain and constipation; rarely black stools in the absence of iron therapy. It is an exceptional case where a child exhibits a lead line along the gums. The most frequent hematological indication is the presence of microcytic, hypochromic anemia. Cerebral involvement may vary from undue drowsiness to deep coma or repeated

grand mal seizures. Sometimes the first clues are repetitive falling, clumsiness and ataxia. A history of pica, or the unusual ingestion of unusual substances, should alert the physician to the possibility of lead poisoning (1, 121, 122, 123, 124).

King (125) gives 300 micrograms of lead from all sources as the maximum daily permissible intake for children. As the average intake increases above this value, the entire amount cannot be excreted, resulting in accumulation within the body. The bodily concentration increases progressively as long as undue ingestion continues. The maximum daily permissible intake was based on caloric intake of young children, air exchange, and on data available on levels of lead in blood of non-exposed and exposed children, including those with frank lead poisoning; results of experimental lead ingestion by adults; fecal lead output in children; initial biologic effects of increased lead intake as related to blood lead levels; rates of increases in lead in the blood of exposed children, and sequelae of lead poisoning.

D. Laboratory Tests

Current emphasis in literature might lead one to believe that laboratory procedures had replaced clinical examination and epidemological investigation. Among the many laboratory aids for the diagnosis of chronic lead poisoning are urine leads, blood leads, glycosuria and occasionally aminoaciduria, fluorescene of free erythrocyte protoporphyrin, increased urinary excretion of coproporphyrins, basophilic stippling of erythrocytes and anemia (241). Findings of more than 50 micrograms of lead per 100 milliliters of whole blood or 240 micrograms per liter of

urine (24-hour specimen) denote actual occurrence of dangerous absorption of lead (242). The upper limits of "safe" values in terms of mean concentrations of lead in the urine are given as a range of 100-240 micrograms of lead per liter of urine. The upper limits of "safe" values for blood have been given as 40-80 micrograms of lead per 100 milliliters of whole blood (241). The upper limits of "safe" values for blood in the pediatric population have been given as 40 micrograms of lead per 100 milliliters of whole blood.

1. Urine Lead

Kehoe (120) also defines the chemical signs of lead absorption. There is first an elevation of the lead concentration in the urine above the ranges of values considered normal for the population at large from 20 to 100 micrograms per liter of urine. The lead urine elevation is followed by an elevation of lead concentration in the blood above the normal range of nine to 50 micrograms per 100 milliliters of blood. In general, a 24-hour sample of urine is preferable to a single sample (even when corrected by conventional means), for a chemical determination of lead. The concentration of lead in the urine is dependent upon a number of variables. Urine usually undergoes relatively prompt changes relative to lead exposure. There are other factors which influence the rate of urinary excretion of lead such as dilution by the amount of water available for excretion and the amount of physical exertion performed by the individual during the collection period. A seasonal factor is also discernible (242). Appropriate urine samples to be representative, therefore, must be at least 24-hour samples. The upper limits of safety for the concentration

of lead in the urine vary significantly with the samples obtained. Individual analysis of lead in urine in excess of 240 micrograms per liter of sample justifies some concern that dangerous absorption of lead may have occurred. Maldonado and Ramos (126) have found that urine concentrations of lead are more useful in diagnosing chronic cases and concentrations of lead in the blood are more indicative in acute cases.

2. Blood Lead

Kehoe has found the severity of the occupational exposure to be more precisely related to the concentration of lead in the blood than in the urine. The concentration of blood lead is considered more useful than that in urine, because blood is not subject to large fluctuations in water content, as urine, or to the influence of changes in renal excretory capacity. However, it should be noted that the correlation between concentrations of lead in blood and that in other tissues of the same person have not been defined (9). The abnormal but safe range in adults varies from 50 to 70 micrograms per 100 milliliters of blood. Because of the increased sensitivity of children and their apparent higher risk for environmental lead absorption, 40 micrograms per 100 milliliters of blood should be the very maximum range for normal in children. In a study of 90 cases of fatal lead encephalopathy, all but one were associated with blood lead concentrations of 150 micrograms per 100 milliliters of whole blood or greater (1). Likewise, symptoms compatible with acute lead poisonings, with few exceptions, were found in children with concentrations greater than 100 micrograms per 100 milli-

liters of whole blood. Kehoe (1) reports that clear-cut symptoms of acute lead poisoning are associated with a blood content greater than 80 micrograms per 100 milliliters of whole blood. Clinical studies substantiate Kehoe's position indicating that clear-cut clinical signs and symptoms of acute lead poisoning are related to the degree of current and recent absorption of lead and, in the absence of severe anemia, are almost always associated with blood lead concentrations greater than 80 micrograms per 100 milliliters of whole blood (9). At higher concentrations than 80 micrograms per 100 milliliters of whole blood, the severity of acute clinical manifestations is not closely associated with and cannot be predicted precisely from the blood lead concentrations. Derangement of heme synthesis is always evident with the risk of symptomatic illness increasing markedly as the blood lead content rises. Cases of lead intoxication have been reported where the blood lead levels never exceeded 50 micrograms per 100 milliliters of blood (121, 127, 128). At the other extreme, a man employed in a silver recovery process plant where lead was used as substrate, on a routine examination was found to have a blood lead level of 1,000 micrograms per 100 milliliters of whole blood. Otherwise, he was generally asymptomatic (129). These cases, however, tend to be the rare exceptions rather than the rule, but they point out the fact that few diagnostic procedures are completely infallible.

3. Other Laboratory Tests

Further laboratory procedures to assist in the diagnosis of lead poisoning are delta-aminolevulinic acid in urine, urinary coproporphyrin, checks for anemia, stippling of red blood cells, and reticulocytosis (116).

None of the signs or clinical pathology can alone confirm or rule out the possibility of lead poisoning.

E. Control of Lead Hazards--Sources of Lead Poisoning (Cases)

1. Industrial Poisoning

a. Scrap Lead Reclamation

Comparative studies have shown that the greatest dangers are in scrap metal salvage, shipbreaking and manufacture of batteries (127, 130, 131, 132, 133, 134, 135, 136, 137). In contemporary scrap metal and shipscrapping industries, the dangers of lead are not as controlled nor as familiar as would be liked. The often chaotic work environment further exacerbates the situation. Too often, lead reclamation is carried out by small junk dealers, poorly financed, under-equipped, and unfamiliar with lead hazards. In Finland, for example, in 1971 twelve of 51 reported cases of clinical lead poisonings were due to scrap smelting. The fact that there were less than 50 workers involved in scrap smelting makes the statistics more impressive (138). Lead reclamation from discarded batteries follows the same course, with the greatest danger of lead intoxication occurring when batteries are melted (139, 140, 141, 142, 143, 144, 145). The dangers of lead intoxication by exposure to lead vapors and ingestion of lead dust have been well documented. With proper worker precautions, properly installed ventilation, and adequate dust and fume collection in our smelters, foundries and other lead plants, and the use of respirators in trades where dusts and fumes cannot be sufficiently contained, such as cutting red lead painted steel, industrial lead poisoning could be eliminated.

b. Tetraethyllead

Tetraethyllead is probably one of the most poisonous products which has one of the best records for rarity of illness associated with its handling and distribution. Certainly, this is true in relationship to its toxicity and total volume produced and used annually. In the United States and Canada, since the introduction of lead as a motor gasoline additive, there have occurred 88 cases of tetraethyllead intoxication, 16 of whom terminated fatally (145). The dangers from tetraethyllead are: cleaning and repairing tanks for the storage of gasoline; processing within the manufacturing plant; handling of tetraethyllead in refineries; and transportation of antiknock compounds (146, 147, 148, 149). Exceptional circumstances which have led to poisoning have ranged from ingestion of the material by accident to ingestion with suicidal intent, its uses as an insecticide, excessive spillage and evaporation of leaded gasoline in fully enclosed and unventilated working areas, and cleaning greasy metal parts with the leaded gasoline (150).

c. Plastic Manufacture

In recent years there has been a continued decrease in the number of occupational lead poisonings. The notable exception to this trend has been the plastic industry where lead stearate is used in the production of polyvinyl chloride plastic (131, 151, 152, 153, 154, 155, 156, 157, 158, 159). Lead stearate is an organic compound $Pb(C_{18}H_{35}O_2)_2$ which comes in the form of a white powder of extremely small particles, and is used as a stabilizer in the manufacture of the plastics. The powder easily builds up dust clouds. It is used in making water pipes, electric cables, rug padding, gutters, and drainpipes.

d. Others

Other cases of lead poisonings have been attributed to: the manufacture and application of leaded paints (160, 161, 162, 163, 164); cutting painted steel girders using oxyacetylene torches causing a massive exposure to lead fumes (165, 166, 167, 168); lead fumes from both smelting and refining furnaces in foundries (129, 169); use of leaded paint in badge enameling (142); work in a pewter factory, polishing the objects with sand paper and abrasive paste (170); lead fumes from traffic inside the Mont Blanc Tunnel--a tunnel worker employed to direct traffic in the 11.7 kilometer tunnel in Chamonix, France (171); tile manufacturing (131, 172); manufacturing of ceramics (173); soldering (174, 175, 176); work inside printing shops (177, 178); restoration of old lead statues (179); and plumbing (180). In most cases of lead poisonings the worker either didn't understand the risk or failed to take the necessary precautions. This is also a failure of management, which has not informed the worker, nor provided the techniques, equipment, training and supervision necessary for prevention. Perhaps, also because of the insidious character of lead intoxication, it is difficult for many workers to realize the inherent dangers of lead fumes and dust causing them to relax their hygienic precautions.

2. Non-Occupational Lead Poisoning (Adults)

a. Alcoholic Beverages

The most frequent cause of adult non-occupational lead intoxication is caused by drinking "moonshine" whiskey (136, 139, 181, 190). In ancient times lead was intentionally added to wines to retard fermentation or to soften unpleasant tastes. As early as 1437 ordinances were

passed to prevent "sweetening" of wine with lead. Current problems with alcohol and lead are due to the fact that lead solder or lead pipes, or both, are used in moonshine stills, added to the fact that discarded automobile radiators, soldered with lead, are used as condensers (191). In 1965, 89 percent of the analyzed samples of Alabama moonshine contained lead, 39 percent being greater than 1,000 micrograms per liter. The difficulty with lead intoxication in alcoholics is differentiating the symptoms due to lead from neurologic manifestations due to alcoholism. It is very easy to overlook lead poisoning on diagnosis of alcoholic myopathy, delirium tremors or alcoholic seizures.

b. Food and Beverage

The second most frequent cause of adult non-occupational lead intoxication is due to contamination of drink or food prepared or stored in containers containing lead. The literature abounds with isolated incidents of lead intoxication due to contaminated food or drink (180). A sampling of cases are: water and wine kept in lead-glazed pottery (192, 195), a reddish-brown jug used to serve cider (196, 197), wine casks coated with amalgam and joined together by copper pipes soldered with tin plate (198), wine made in lead-glazed bathtubs (179, 199, 200), a wine barrel painted with minium to prevent rust (201), food kept in lead-glazed pots (202, 203, 204, 205), automatic machine washing of cocktail glassware which caused the dissolution of the white-leaded inside frosting (206), lead-lined drinking glasses (207), dinnerware (208), and salt from an antique salt shaker composed

of 80 percent lead (209). This list is by no means complete, but hopefully it serves to indicate the potential contamination of food and drink from containers that can leach lead. It would appear that the greatest risk items are alcoholic beverages and acidic type foods. The consumers, unaware of the proliferation of products containing lead on the market or of lead concentrations in products they use, can unknowingly ingest sufficient quantities of lead to cause poisoning. Many people, apparently, are not aware of the problems that lead can cause, or do not realize the proliferation of lead products.

c. Potable Water

Other cases of non-occupational lead intoxication poisonings have been reported as due to water. In one case the water was contaminated from lead joints within the water softener (210). A more commonly reported type of water contamination is from water allowed to stand in lead storage tanks or lead pipes (211).

d. Others

Other sources for lead poisoning cases have been: barley water for cystitis prepared in a lead jug (213), an ointment used to prevent baldness that contained lead (213), "sindoor" (a red powder applied to the face of married women)--thought to have been ingested (214), and lead powder from a gunshot wound (215).

3. Pediatric Poisoning

As lead in the workplace became more stringently controlled, the emphasis on lead poisoning shifted from the industrial setting to the

community where the public was alerted to the potential ills of lead in the environment. Almost without exception the victims of environmental lead poisoning have been children, usually children from one to four years of age. The major cause of environmental lead poisoning has been, and continues to be, lead-based paint. White lead, basic lead carbonate, was in the past extensively used as a pigment for paints. The widespread use of white lead paints in the past, both indoors and outdoors, means there are many old buildings in which these paints can still be found, even though they may have been covered with other paints. In addition to lead in paint, there is also a danger from lead-contaminated street dust and soil along main thoroughfares. Existing evidence indicates that lead concentrations of contaminated city soil may be 20,000 parts per million (34), while polluted city air may contain up to 9.0 micrograms of lead per cubic meter of air (31, 38). As the source of environmental lead poisoning differs from industrial lead poisoning, so do the groups affected differ.

In contrast to adult occupational lead poisoning where the intoxication results from prolonged exposure to air with excessive lead content with the condition usually being chronic and the rate of deterioration of biological functions slow, lead poisoning in children proceeds at a swift pace and the response of symptoms can be correlated to lead poisoning following lead ingestion or overwhelming exposures of lead concentrations such as battery salvage and tetraethyllead vapor (241). The clinical signs and symptoms develop rapidly, usually within three months following the beginning of excessive lead ingestion, and death can also occur rapidly (216). The biological differences between adults and children

are anatomic, physiologic, pathologic, and immunologic, with body weight, height, caloric intake, basal metabolic rate, nutritional disorders, resistance, etc. performing differently.

a. Pica

Nearly all cases of pediatric lead poisonings have been attributed to pica and ingestion of old paint, plaster, caulking chips or putty containing lead (1, 9, 95, 96, 97, 98, 181, 217, 218, 219, 220, 221, 222, 223, 224, 225, 226, 227, 228, 229, 230, 231, 232). Lead-based paint has been found to contain as much as 300,000 micrograms of lead per gram of paint (9). It is estimated in the United States that 600,000 children annually have increased lead absorption, 25 percent of whom may suffer permanent neurologic damage. King estimates that 300 micrograms of lead is the maximum daily intake of lead without excessive body lead burden in children. The daily intake of lead by urban children from food and water ranges from 106-146 micrograms (125).

Ingestion of sufficient quantities of paint flakes, or street or house dirt, containing lead can cause the total intake to exceed the maximum permissible daily intake of 300 micrograms and result in blood lead elevation and can eventually progress into lead intoxication.

Although great progress has been made in recent years to prevent and control the absorption of lead by both adults and children, there are still some children, particularly those residing in city slums, who are still poisoned from eating leaded paint applied to old buildings over 30 years ago. Nearly everyone agrees that the prime source of lead intoxication in children is old lead-based paint and that lead intoxication

in children is preventable. The Lead Industries Association has compiled seven steps to prevention:

> "(1) Alert and warn parents and others who live in dwellings which have lead paint in interiors;
>
> (2) Remove sources of lead that children can eat;
>
> (3) Take steps to keep any child suspected of eating lead from further exposure;
>
> (4) Physicians, public health nurses, and others should watch for early symptoms of lead absorption;
>
> (5) Quick and accurate diagnosis prevents serious consequences;
>
> (6) Proper and careful treatment should start immediately after diagnosis of lead intoxication;
>
> (7) When a case is found, check other children in the home immediately for possible signs of lead absorption." (232)

b. Other Pediatric Sources

Because lead poisoning in children is most often due to ingestion of plaster and chips of lead-based paint, there is often the tendency to think of lead poisoning occurring only in children who live in dilapidated housing where the deteriorated conditions and pica act as co-culprits. However, there are reports in the literature of pediatric lead poisonings due to other sources. Cases of chronic lead poisoning in children are reported to have been caused by intoxication from: lead dust from smelters when children lived near the lead works (233, 234); lead fumes caused by the extraction of gold and silver from jewelers' wastes, an occupation practiced in the homes by the parents (141, 214, 235, 236); fumes from the burning of battery cases (141, 181); and home battery manufacture (181). Other sources of lead have been attributed to leaded paint on toys and

furniture, lead nipple shields, a lead washer, baubles, etc. (9, 181, 237). Children, like adults, have also been diagnosed as having contacted lead poisoning from food and drink contaminated from lead-glazed earthenware (180, 192, 193, 194, 195, 202, 203, 204, 205, 206). Along with food and drink being contaminated from lead-glazed earthenware, a report from Africa describes a case of lead poisoning due to contamination of dried milk as it was reconstituted in a mixer that had been repaired with lead solder (238). Another case of lead poisoning was reported of a child chewing magazine paper which had been printed with colored ink containing lead (239). Currently there is an increasing trend among middle class families to return to the inner city and to buy older dwellings and restore them. Two cases of lead intoxication in unrelated incidents were reported as being due to the inhalation or swallowing of small particles of lead dust generated by the sanding and scraping of the old paint in the process of restoring the dwellings (240).

V SUMMARY

Lead is used in almost every major industry with more than 1.6 million employees in a host of industrial manufacturing processes and in many service industries potentially exposed. Lead is all around us in our environment, in water, air and the soil; it is in every living thing and in many of the products with which man comes into contact. Lead can enter the body by inhalation or by ingestion and, in the case of organic lead, by absorption through the skin. In sufficient quantities, the result is the same--severe gastrointestinal, blood, and central nervous system disorders.

Inhalation of lead dust or fumes has been the most frequent means of entry and has resulted in most of the industrial health problems encountered with this metal. Lead is a cumulative poison; a part of a small daily dose is not eliminated, but is stored in the body. Eventually, a point is reached where symptoms and disability--even death--occur. A particular measurement of lead in the environment reflects some unknown fraction that might come into effective contact with man and have the potential for causing a biological disturbance. Lead is a known toxic substance for which no beneficial biological role has been demonstrated. Experimental evidence suggests that the least measurable quantities of lead within the cellular structure are capable of affecting cellular metabolism and these effects are a function of lead concentration. In the not too distant past, poisoning clearly attributable to airborne lead exposure was a serious and frequently encountered disease among workers in lead smelters and in the lead-using industries. Today,

industrial hygienic programs and advanced diagnostic procedures have substantially reduced the hazard to lead, enough so that it can be said that industrial lead intoxication is a vanishing malady. However, workers may be occupationally exposed to hazardous concentrations of lead by cutting red lead painted steel. Also, potentially dangerous conditions may still exist in small shops, particularly those involved in lead reclamation, where supervision is minimal and occupational health programs are non-existent.

If the lead industries are excluded, then the only hazardous circumstance relative to atmospheric lead pollution in man is the urban setting. The high concentrations of lead in urban air and on vegetation and streets constitute a source of intake, additional to the usual dietary sources, and in special circumstances may be a substantial source. Although the concentrations of lead in the air of cities pose no threat to the general population, the extent to which it increases lead absorption and lead poisoning in young children is not known but thought to be very small. With respect to young children, it is not a matter exclusively of inhalation and particle size, inasmuch as very young children mouth and eat things that are not food rather indiscriminately. The prevalence of pica in young children complicates the problem. Airborne lead wastes from such sources as automotive emission and the weathering and demolition of old buildings can be expected to have a significant effect on the total intake. Regardless of extraneous sources, the direct ingestion of lead-pigment paints is undoubtedly the principal environmental source in cases of severe acute lead poisoning in young children.

Lead ingested by young children in pica is almost entirely due to lead-pigmented paint applied over 30 years ago. The traditional white lead paint weathered by chalking and so some of the lead carbonate would have been washed away by rain, to be dispersed into the environment. Lead paints constitute a recognized hazard because of flaking and chalking, and from the lead-containing particles released when old painted timber and painted furniture are burned. On the positive side, leaded paint production has declined sharply from the average of over 120,000 tons of lead per year in the 1920's to only 2,440 tons in 1974. The Government restricted the amount of lead that may be contained in paint to 0.5 percent in 1970. There has been concern by the Consumers Union that this level is inadequate and they are lobbying for a maximum level of 0.06 percent lead in paint, the same level the Childhood Poisoning Prevention Committee advocates. It is hoped that within a few years the problem of lead-pigmented paint will have disappeared as older structures are torn down and destroyed.

Following the incidence and severity of lead poisoning in young children dwelling in deteriorated houses and urban slums and the industrial lead workers, lead intoxication has been most frequently seen in those persons who imbibe illicit whiskey regularly. Isolated cases of lead poisoning occur from food and drink prepared in leaded containers. Other sources of lead intoxication are rare.

Insidious lead poisoning is more prevalent than is generally realized. It is not easily diagnosed and unfortunately is often missed. Early diagnosis is imperative, and it would be hoped that knowledge of the ubiquitous lead products which have led to lead intoxication in the past, along with

the intricacy of lead intoxication symptomatology would be helpful in aiding the diagnostician in the absence of adequate histories.

VI BIBLIOGRAPHY

1. Kehoe, R. A.: THE METABOLISM OF LEAD IN MAN IN HEALTH AND DISEASE: THE HARBEN LECTURES, 1960. J. Roy. Inst. Public Health and Hyg., 24: 81-97, 101-120, 129-143, 177-203, 1961.

2. de Treville, R. T. P.: NATURAL OCCURRENCE OF LEAD. Arch. Envir. Health, 8:212-221, 1964.

3. Kehoe, R. A.: METHODS FOR THE PREVENTION OF LEAD POISONING IN INDUSTRY. Journal of Occupational Medicine, 6(6):287-294, 1964.

4. United States Department of Interior, Bureau of Mines: MINERAL FACTS AND PROBLEMS. U. S. Government Printing Office, Wash., D. C., 1970, pp. 603-620.

5. National Security Resources Board: MATERIALS SURVEY LEAD. U. S. Government Printing Office, Wash., D. C., May 1951, pp. I-3-4.

6. Lead Industries Association, Inc: ANNUAL REVIEW U. S. LEAD INDUSTRY, 1974. New York, 1975, p. 6.

7. Bureau of Mines: MINERALS YEARBOOK. U. S. Government Printing Office, Wash., D. C., 1872-1975.

8. Ziegfeld, R. L.: IMPORTANCE AND USES OF LEAD. Arch. Envir. Health, 8:202-212, 1964.

9. Committee on Biological Effects of Atmospheric Pollutants, National Academy of Sciences: AIRBORNE LEAD IN PERSPECTIVE. National Academy of Science, Wash., D. C., 1972.

10. Hendrick, K. G.: LEAD "THE GYNOCRATIA METAL". Lead Industries Assoc., Inc., New York, 1975.

11. Hepple, P.: LEAD IN THE ENVIRONMENT. Applied Science Publishers, Ltd., Barking, Essex, England, 1972, pp. 3-6.

12. Casarett, L. J. and Doull, J.: TOXICOLOGY, THE BASIC SCIENCE OF POISONS. Macmillan Publishing Co., Inc., New York, 1975, p. 481.

13. McFeatters, A.: A BATTLE THAT WON'T STAY WON. Cincinnati Post, Cincinnati, Ohio, Jan. 8, 1976, p. 14.

14. Barry, P. S. I. and Harrison, G. F. (The Associated Octel Co., Ltd., London, England): MOTOR GASOLINE, LEAD AND AIR POLLUTION. Petroleum Review, 26:311-318 (Oct), 1972.

15. Cholak, J., Schafer, L. J., and Yeager, D.: THE AIR TRANSPORT OF LEAD COMPOUNDS PRESENT IN AUTOMOBILE EXHAUST GASES. Amer. Ind. Hyg. Assoc. Journ., 29:562-568, 1968.

16. Mitchell, D. G. and Aldous, K. M.: LEAD CONTENT OF FOOD STUFFS. Env. Health Perspectives, Experimental Issue, 7:59-64, 1964.

17. Kolbye, A. C., Jr., Mahaffey, K. R., Fiorino, J. A., Corneliussen, P. C., and Jelinek, C. F.: FOOD EXPOSURE TO LEAD. Env. Health Perspective, 7:65-74, 1974.

18. Bertinuson, J. R. and Clark, C. S.: THE CONTRIBUTION TO LEAD CONTENT OF SOILS FROM URBAN HOUSING. Interface, 2:6, 1973.

19. Bogden, J. D., Joselov, M. M., and Smith, N. P.: EXTRACTION OF LEAD FROM PRINTED MATTER AT PHYSIOLOGICAL VALUES OF PH. Env. Health, 30:442-444, 1975.

20. Lead Industries Association: LEAD IN MODERN INDUSTRY. Lord Baltimore Press, 1952, pp. 127-137.

21. Hamilton, A.: THE PREVALENCE OF INDUSTRIAL LEAD POISONING IN THE UNITED STATES. From Medicine Monographs Vol. VII, Lead Poisoning, The Williams & Wilkins Co., Baltimore, Md., 1926, pp. 232-240.

22. National Safety Council: TARGET HEALTH HAZARD: LEAD. National Safety News, 108:72-78 (Nov), 1973.

23. Patterson, C. C.: CONTAMINATED AND NATURAL LEAD ENVIRONMENTS OF MAN. Arch. Envir. Health, 11:344-346, 1965.

24. Chow, T. J. and Patterson, C. C.: THE OCCURRENCE AND SIGNIFICANCE OF LEAD ISOTOPES IN PELAGIC SEDIMENTS. Geochem, Cosmochem, Acta, 26:263-308, 1962.

25. Blanchard, R. L.: RELATIONSHIP BETWEEN POLONIUM-210 AND LEAD-210 IN MAN AND HIS ENVIRONMENT. In Aberg, B. and Hangate, F. P., Eds., "Proceedings of the International Symposium on Radiological Concentration Process," New York, Pergama Press, 1966, pp. 281-294.

26. Goldschmidt, V. M.: GEOCHEMISTRY. Edited by Alex Muir, Oxford, Clarenden Press, 1954.

27. Kehoe, R. A., Thamann, F., and Cholak, J.: ON THE NORMAL ABSORPTION AND EXCRETION OF LEAD: I, II, AND III. J. Industr. Hyg. 15:257-300, 1933.

28. Connor, J. J. and Shacklette, H. T.; with sections on Field Studies by Ebens, R. J., Erdman, J. A., Miesch, A. T., Tidball, R. R., and Tourtelot, H. A.: BACKGROUND GEOCHEMISTRY OF SOME ROCKS, SOILS, PLANTS, AND VEGETABLES IN THE CONTERMINOUS UNITED STATES. U. S. Geological Survey Professional Paper 574, 1975, p. 168.

29. National Research Council: AIRBORNE LEAD IN PERSPECTIVE. National Academy of Science, Wash., D. C., 18:26, 27, 30, 37, 1972.

30. Huff, L. C.: ABNORMAL COPPER, LEAD AND ZINC CONTENT OF SOIL NEAR METALLIFEROUS VEINS. Econ. Geol., 47:517-542, 1925.

31. Motto, H. L., Daines, R. H., Chilko, D. M., and Motto, C. K.: LEAD IN SOILS AND PLANTS: ITS RELATIONSHIP TO TRAFFIC VOLUME AND PROXIMITY TO HIGHWAYS. Envir. Sci. & Tech., 4:231-238, 1970.

32. Cholak, J., Schafer, L. J., and Sterling, T. D.: THE LEAD CONTENT OF THE ATMOSPHERE. APCA Journal, 11:231-238, 1970.

33. Ewing, B. B. and Pearson, J. E. (Univ Illinois at Urbana-Champaign, Urbana): LEAD IN THE ENVIRONMENT. In Pitts, J. N., Jr., Metcalf, R. L., and Lollyd, A. C., Ed.: Advances in Env. Sc. & Tech., New York, John Wiley & Sons, 1974, Vol. 3, pp. 1-127.

34. Baltrop, D. (St. Bartholomew's Hosp. Med. Coll., London, England): CHILDREN AND ENVIRONMENTAL LEAD. From: Lead in the Environment, Ed.: Hepple, P., Barking, Essex, England, Applied Science Publishers, Ltd., 1972, pp. 52-60.

35. Cholak, J., Schafer, L. J., and Hoffer, R. F.: COLLECTION AND ANALYSIS OF SOLIDS IN URBAN ATMOSPHERES. Arch. Ind. Hyg. Occupational Med., 2:443, 1950.

36. Chambers, L. A., Foter, M. J., and Cholak, J.: A COMPARISON OF PARTICULATE LOADINGS IN THE ATMOSPHERES OF CERTAIN AMERICAN CITIES. Proceedings Third National Air Pollution Symposium, Pasadena, Calif., 1955, pp. 24-32.

37. Lawther, P. J., Commins, B. T., Ellison, J. McK., and Giles, D.: AIRBORNE LEAD AND ITS UPTAKE BY INHALATION. From: Lead in the Environment, Ed.: Hepple, P., Applied Science Publishers, Ltd., Barking, Essex, England, 1972, pp. 8-28.

38. Katsunuma, H.: SEVERAL QUESTIONS ON THE HUMAN BODY AND LEAD. Rodono Kagaku (Dig. Sci. Labor.), 28:4-10, 1973.

39. Resch, W. and Hauck, H. (Vienna, Austria): COLLOQUIUM: ENVIRONMENTAL POLLUTION AS A STRESS FACTOR. Staub Reinhalt ung der Luft in English, 33:231-232, (May), 1973.

40. Byalko, N. K., and Chulina, N. A.: BILIRUBINEMIA IN LEAD POISONING. O bilirubinimii pri intoksikatsii svintsom. In: Letavet, A. A., Ed.: Occupational Diseases in the Chemical Industry. Professional' nye zabolevaniya v khimicheskoi promyshlennosti. Moscow, Meditsina, 1965, pp. 191-194.

41. Epstein, S. S., Chairman (Harvard Med. School, Boston, Mass.): TOXICOLOGICAL AND EPIDEMOLOGIC BASES FOR AIR QUALITY CRITERIA. Journal of the Air Pollution Control Association, 19:629-732, (Sept.), 1967.

42. Snodderly, D. M., Jr. (The Retina Foundation, Boston, Mass.): BIOMEDICAL AND SOCIAL ASPECTS OF AIR POLLUTION. In Pitts, J. N., Jr., Metcalf, R. L., and Lloyd, A. C., Ed.: Advances in Environmental Science and Technology, New York, John Wiley & Sons, 1974, Vol. 3, pp. 157-266.

43. Golz, H. H. (Washington, D. C.): EPA'S POSITION ON THE HEALTH EFFECTS OF AIRBORNE LEAD. A CRITIQUE. Journal of Occupational Medicine, 15:369-373, (Apr.), 1973.

44. Kirby, G. F. and Gottwald, F. D.: LEAD AND AIR POLLUTION: AN ANALYSIS OF THE MORSE REPORT. Ethyl Corp., Aug, 1970,

45. Kehoe, R. A.: CONTAMINATED AND NATURAL LEAD ENVIRONMENTS OF MAN. Archives of Environ. Health, 11:736-739, (Nov.), 1965.

46. Hardy, H. L., Chamberlin, R. I., Maloof, C. C., Boylon, G. W., Jr., and Howell, M. C. (Mass. Inst. Tech., Cambridge): LEAD AS AN ENVIRONMENTAL POISON. CLINICAL PHARMACOLOGY AND THERAPEUTICS. 12:982-1002, (Nov.-Dec.), 1971.

47. U. S. Environmental Protection Agency, Office of Research and Monitoring: EPA'S POSITION ON THE HEALTH EFFECTS OF AIRBORNE LEAD. Washington, D.C., Nov. 29, 1972.

48. Bernhart, A. P. (Univ. Toronto, Canada): NOTES ON AIR POLLUTION: THE INGREDIENTS DEFINED; METEOROLOGY AND PLANNING. Water and Pollution Control (Toronto), 109:41-42, (Oct.); 57-58 (Nov.), 1971.

49. Kehoe, R. A. (Univ. of Cincinnati, Ohio): CONTAMINATED AND NATURAL LEAD ENVIRONMENT OF MAN. (Letter to the Editor), Archives of Environmental Health, 11:736-739, (Nov.), 1965.

50. Schroeder, H. A. (Darmouth Med. School, Brattleboro, Vermont): (A Letter to the Editor on Contaminated and Natural Environment of Man by Patterson, C. C.). Archives of Environmental Health, 12:270-271, (Feb.), 1966.

51. Danielson, L.: GASOLINE CONTAINING LEAD. Swedish Natural Science Research Council, Ecological Research Committee, Bulletin No. 6, 1970.

52. Hicks, R. M. (Middlesex Hosp. Med. Sch., London, England): AIR-BORNE LEAD AS AN ENVIRONMENTAL TOXIN, A REVIEW. Chemico-Biological Interactions 5, No. 6:361-390, 1972.

53. Farkas, E. J. (McGill Univ., Montreal, Canada): LEAD IN GASOLINE: A SKEPTICAL REVIEW OF FAVORABLE CLAIMS. Water and Pollution Control, 110:40-42, (June), 1972.

54. Needleman, H. L. and Scanlon, J. (Children's Hosp. Med. Ctr., Boston, Mass.): GETTING THE LEAD OUT. New England Journal of Medicine, 288:466-467, (Mar. 1), 1973.

55. Schuck, E. A. (Univ. California, Riverside): AIR AND LAND POLLUTION. Professional Sanitation Management, 4:17-26, (Apr.), 1972.

56. Snyder, R. B., Wuebbles, D. J., Pearson, J. E., and Ewing, B. B. (Illinois Inst. Environ. Quality, Chicago): A STUDY OF ENVIRONMENTAL POLLUTION BY LEAD. U. S. National Technical Information Service Report PB 205 239, 1971.

57. International Labour Office: WHITE LEAD. P. S. King & Son, Ltd., London, 1927, pp. 26-38.

58. Kehoe, R. A., Cholak, J., and Story, R. V.: A SPECTROCHEMICAL STUDY OF THE NORMAL RANGES OF CONCENTRATION OF CERTAIN TRACE METALS IN BIOLOGICAL MATERIALS. J. Nutr, 19:579-592, 1940.

59. Kehoe, R. A., Cholak, J., and Largent, E. J.: THE HYGIENIC SIGNIFICANCE OF THE CONTAMINATION OF WATER WITH CERTAIN MINERAL CONSTITUENTS. J. Amer. Water Works Assn., 36:645-657, 1944.

60. Hem, J. D., and Durum, W. H. (U. S. Dept. Interior, Menlo Park, Calif.): SOLUBILITY AND OCCURRENCE OF LEAD IN SURFACE WATER. J. Amer. Water Works Assn., 65:562-568, 1973.

61. Monier-Williams, G. W.: LEAD IN FOOD REPORTS. Public Health Medical Subjects, Ministry of Health, London, No. 88, 1938.

62. Goldberg, A.: DRINKING WATER AS A SOURCE OF LEAD POLLUTION. U. S. National Institute of Env. Health Sc. and U. S. Env. Protection Agency, Low Level Lead Toxicity, Env. Health Perspectives Experimental Issue No. 7:103-105, 1974.

63. Aksyuk, A. F., and Boldina, Z. N. (USSR): (HYGIENIC EVALUATION OF HIGH-IMPACT VINIPLAST). Gig. Vop. Proizvod. Primen. Polim. Mater. 1969:155-158, Mosk. Nauch.-Issled. Inst. Gig.: Moscow, USSR.

64. Packham, R. F. (Water Res. Assoc., England): THE LEACHING OF TOXIC STABILIZERS FROM UNPLASTICIZED PVC WATER PIPE: PART I - A CRITICAL STUDY OF LABORATORY TEST PROCEDURES; PART II - A SURVEY OF LEAD LEVELS IN u PVC DISTRIBUTION SYSTEMS; PART III - THE MEASUREMENT OF EXTRACTABLE LEAD IN u PVC PIPES. Water Treatment and Examination 20, Part 2: 108-124; Part 3:144-151; 152-164, 1971.

65. Bogen, D. C. (New York Operations Office (AEC), N. Y. Health and Safety Lab.): STABLE LEAD CONCENTRATIONS IN NEW YORK CITY FOODSTUFFS. U. S. Atomic Energy Commission Document No. HA 56-246, 1972, pp. I 45-52.

66. Great Britain, Ministry of Agriculture, Fisheries and Food: SURVEY OF LEAD IN FOOD. Working Party on the Monitoring of Foodstuffs for Heavy Metals: Second Report, London, H. M. Stationery Office, 1972.

67. Shacklette, H. T., Erdman, J. A., and Keith, J. R. (U. S. Geol. Survey, Denver, Colo.): TRACE ELEMENTS IN UNPROCESSED PLANT FOODSTUFFS. In Savage, E. P., Ed.: Environmental Chemists Human and Animal Health, Proceedings of 2nd Annual Conference, Colorado State University, Ft. Collins, July, 1973, pp. 129-144.

68. Warren, H. V. (Univ. British Columbia, Vancouver, Can.): VARIATIONS IN THE TRACE ELEMENT CONTENT OF SOME VEGETABLES. Jour. Royal Col. Gen. Prat. 22, No. 114:56-60, 1972.

69. Warren, H. V. and Delavault, R. E. (Univ. British Columbia, Vancouver, Can.): VARIATIONS IN THE COPPER, ZINC, LEAD AND MOLYBDENUM CONTENTS OF SOME VEGETABLES AND THEIR SUPPORTING SOILS. In Cannon, H. L. and Hopps, H. C., Ed.: Environmental Geochemistry in Health and Disease, Geol. Soc. Amer., Memoir 123, 1971, pp. 97-108.

70. Thomas, B., Roughan, J. A., and Watters, E. D. (Min. Agric., Fisheries and Food, Hatching Green, Harpenden, Herts, England): LEAD AND CADMIUM CONTENT OF SOME VEGETABLE FOODSTUFFS. Jour. Sc. Food & Agric. 23 No. 12:1493-1498, 1972.

71. Ruff, H. (Fed. Chem. Res. Inst. Erlangen, W. Ger.): Bedarfsgegenstaende aus keramischen Massen, Glasuren und Emaille in lebensmith elchemischer sicht. (UTENSILS MADE OF CERAMICS, GLAZES AND ENAMEL VARNISHES FROM THE FOOD CHEMISTRY POINT OF VIEW). Zeitschrift fuer Lebensmittel - Untersuchung und-Forschung 149, No. 5:284-289, 1972.

72. Francis, C. W., Chesters, G. and Erhardt, W. H.: POLONIUM - 210 ENTRY INTO PLANTS. Environ. Sc. Tech. 2:690-695, 1968.

73. Horak, O., and Huber, I. (Austrian Soc. Res. At. Energy, Vienna, Austria): CONTAMINATION OF PLANTS AND SOILS BY LEAD RESIDUES FROM PETROL ENGINES. Ber. Oesterr. Studienges. Atomenerg, 1973, SGAE BER. No. 2077.

74. Page, A. L., Ganje, T. J., and Joshi, M. S. (Dept. Soil Sci. Agric. Eng., Univ. California, Riverside): LEAD QUANTITIES IN PLANTS, SOIL, AND AIR NEAR SOME MAJOR HIGHWAYS IN SOUTHERN CALIFORNIA. Hilgardia 41:1-31, (July), 1971.

75. Nishiyima, M., Veda, K., Maki, T., Wada, M., Takahashi, S., Kimura, Y., Yamada, T., Nakajima, K., and Fukuda, T. (Tokyo Metrop. Res. Lab. Public Health, Japan): (HEAVY METALS DISSOLVED IN CANNED FOODS, II. DETECTION OF TIN AND LEAD FROM VARIOUS CANNED FRUITS AFTER OPENING). Tokyo Toritsu Eisei Kentyusho Kenkyu Nempo, 23:189-196, 1971.

76. Kimura, Y., Ueda, K., Nishizima, M., Maki, T., Wada, M., Takahashi, S., Yamada, T., Arai, M., and Uematsu, T. (Dep. Food Hyg., Tokyo Metrop. Res. Lab. Public Health, Japan): (EXTRACTABLE HEAVY METALS IN CANNED FOODS. I. DETECTION OF TIN AND LEAD FROM VARIOUS SORTS OF CANNED BABY FOODS AFTER THEIR OPENING). Tokyo Toritsu Eisei Kenkyusho Kenkyu Nepo, 22:95-100, 1970.

77. Anonymous: LEAD IN BABY FOODS. Notes and News, Lancet, 2:96, (July), 1972.

78. Murthy, G. K., and Rhea, U. S. (U. S. Food Drug Admin., Cincinnati, Oh.): CADMIUM, COPPER, IRON, LEAD MANGANESE, AND ZINC IN EVAPORATED MILK, INFANT PRODUCTS AND HUMAN MILK. Journal of Dairy Science, 54:1001-1005, (July), 1971.

79. Lamm, S., Cole, B., Glynn, K., and Ullmann, W. (Connecticut State Dept. Health, Hartford): LEAD CONTENT OF MILK FED TO INFANTS - 1971-1972. New England Journal of Medicine, 289:574-575, (Sept. 13), 1973.

80. Great Britain, Ministry of Agriculture, Fisheries and Food: SURVEY OF LEAD IN FOOD. Working Party on the Monitoring of Foodstuffs for Heavy Metals: Second Report, London, H. M. Stationery Office, 1972.

81. Berman, E., and McKiel, K. (Cook County Hosp., Chicago, Ill.): IS THAT TOOTHPASTE SAFE? Archives of Envir. Health, 25:64-65, (July), 1972.

82. Shapiro, I. M., Cohen, G. H., Needleham, H. L., and Tuncay, O. C. (Univ. Pennsylvania, Philadelphia): THE PRESENCE OF LEAD IN TOOTHPASTE. Journal of the American Dental Association, 86:394-395, (Feb.), 1973.

83. Scholl, W. (Fed. Agr. Res. Inst. Augustenberg, Karlsruhe-Durlach, Germany): Blei wird aus der Glasur von Buntgeschirr herausgel ost. (THE LEACHING OF LEAD FROM THE GLAZE OF COLORED EARTHENWARE), Deutsche Medizinische Wochenschrift, 97:438, (Mar. 19), 1972.

84. Steele, E. A. (Bur. Foods, U. S. Food Drug Admin.): ACTIVITIES OF FDA - LEAD IN POTTERY. Speech delivered to American Ceramic Society, Southern California Section and Northern California Section, Feb. 17, 1971, Los Angeles, and Feb. 18, San Francisco.

85. Hickman, J. R.: LEAD POISONING: POTTERY GLAZES, AN OFTEN-IGNORED HAZARD. Presented at the Canada Safety Council, Fredericton, New Brunswick, May 26, 1970. Preprint, Canada Safety Council, Fredericton, New Brunswick, 1970, From Air Pollution Abstracts 3:17741, 1972.

86. Anonymous: APPLE-JUICE COLIC. Lancet, 1:278, (Feb. 6), 1971.

87. Krinitz, B. and Hering, R. K. (FDA): TOXIC METALS IN EARTHENWARE. FDA Papers, 5:21-24, (Apr.), 1971.

88. May, R. (MD Internal Med., Kreuth nr Tegernsee, W. Germany): Blei in Buntgeschirr-Glasur. (LEAD IN COLOR GLAZED CROCKERY). Deutsche Medizinische Wochenschrift, 97:740, (May 5), 1972.

89. Browder, A. A. (Coll. Med. Denistry, New Jersey at Newark): LEAD POISONING FROM GLAZES. Annals of Internal Medicine, 76:665, (Apr.), 1972.

90. American Medical Association Department of Environmental Public and Occupational Health (Chicago): HAZARDS OF LEAD POISONING FROM OLD PEWTER. Questions and Answers, Journal of the American Medical Association, 222:374, (Oct. 16), 1972.

91. U. S. Food and Drug Administration: SILVER-PLATED HOLLOWARE. Federal Register, 40:6523-6524, (Feb. 12), 1975.

92. Ball, G. V. and Morgan, J. M.: CHRONIC LEAD INGESTION AND GOUT. Southern Medical Journal, 61:21-24, 1968.

93. Anonymous: POISON MOONSHINE WHISKEY FACT SHEET. U. S. Treasury Department, Internal Revenue Service Document No. 5305, November, 1960.

94. Lin-Fu, J. S.: LEAD POISONING IN CHILDREN. Social and Rehabilitation Service, U. S. Department of Health, Education, and Welfare, Children's Bureau Publication 452, Washington, D.C., U. S. Government Printing Office, 1963.

95. American Academy of Pediatrics, Committee on Environmental Hazards and Subcommittee on Accidental Poisoning of Committee on Accident Prevention (P. F. Wehrle, Chairman): ACUTE AND CHRONIC CHILDHOOD LEAD POISONING. Pediatrics 47:950-951, (May), 1971.

96. Hankin, L. (Connecticut Agric. Expt. Sta., New Haven): LEAD POISONING - A DISEASE OF OUR TIME. Journal Milk and Food Technology 35, No. 2:86-97, 1972.

97. Ascher, S. F. (Food Drug Admin., New York, N. Y.): HISTORY AND EPIDEMIOLOGY OF LEAD POISONING. Public Health News 51, No. 7:151-152, 1970.

98. De la Burde, B., and Reames, B. (MCR Sta., Richmond, Va.): PREVENTION OF PICA, THE MAJOR CAUSE OF LEAD POISONING IN CHILDREN. American Journal of Public Health 63:737-743, (Aug.), 1973.

99. Anonymous: PENCIL CHEWERS BEWARE. Chemistry 44:5-6, (Nov.), 1971.

100. Schaplowsky, A. F. (Health Serv. Mental Health Admin., Cincinnati, Ohio): LEAD IN PAINT ON PENCILS. HSMHA Health Reports 86:961-962, (Nov.), 1971.

101. Pichirallo, J.: LEAD POISONING: RISKS FOR PENCIL CHEWERS? Science 173:509-510, (Aug. 6), 1971.

102. Schucker, G. W., Vail, E. H., Kelley, E. B., and Kaplan, E.: PREVENTION OF LEAD PAINT POISONING AMONG BALTIMORE CHILDREN: A HARD-SELL PROGRAM. Public Health Rep. 80:969-974, 1965.

103. Joel, L. S., and Berger, H. W.: REPORT ON A PRE-TEST OF A SURVEY PLAN FOR ESTIMATING INCIDENCE OF LEAD BASED PAINT. Final Report, U. S. National Bureau of Standards Report NBSIR 73-407, 1973.

104. Bertinuson, J.: EVALUATION OF LEAD SOURCES IN PEDIATRIC RESIDENTIAL ENVIRONMENT. Thesis, MS, University of Cincinnati, 1973.

105. U. S. Department of Health, Education and Welfare: HEALTH SERVICES REPORT No. 3, 89:301-302, (May-June), 1974.

106. Obata, T., Kano, S., and Yajima, N.: (ON THE CONTENTS OF LEAD IN HAIR SPRAYS). Bulletin of National Institute of Hygienic Sciences (Tokyo), 87:82-84, 1969.

107. Mehani, S.: LEAD RETENTION BY THE LUNGS OF LEAD-EXPOSED WORKERS. Ann. Occupational Hygiene, 9:165-171, 1966.

108. Kehoe, R. A., Thamann, F., and Cholak, J.: LEAD ABSORPTION AND EXCRETION IN CERTAIN LEAD TRADES, Journal Industrial Hygiene, 15:306-319, 1933.

109. Goodwin, T. W., Ed.: BIOCHEMICAL SOCIETY SYMPOSIUM 28: PORPHYRINS AND RELATED COMPOUNDS. New York, Academic Press Inc., 1968.

110. Mayers, M. R.: OCCUPATIONAL HEALTH HAZARDS OF THE WORK ENVIRONMENT. Baltimore, Williams & Wilkins Co., 1969, pp. 68-70.

111. Johnstone, R. T.: CLINICAL INORGANIC LEAD INTOXICATION. Archives Environmental Health, 8:250-255, (Feb.), 1964.

112. Zavon, M. R.: PROBLEMS IN RECOGNITION OF LEAD INTOXICATION. Archives Environmental Health, 8:262-265, (Feb.), 1964.

113. Kayo, S. (Inst, Legal Med., Univ Puerto Rico, Rio Piedras): GENERAL GUIDE TO THE DIAGNOSIS AND TREATMENT OF POISONINGS. Boletin de la Association Médica de Puerto Rico, 62:283-288, (Oct.), 1970.

114. Sverdlov, S. L. (Norozybkor Mun. Hosp., Bryansk Distr., USSR): Differentsial naya diagnostika svintsovoi koliki i ostrykh khirurgicheskikh zabolevanii organov bryushnoi polosti. (DIFFERENTIAL DIAGNOSIS OF COLIC CAUSED BY LEAD POISONING AND ACUTE SURGICAL DISORDERS OF THE ABDOMINAL ORGANS). Klinicheskaya Meditsina, 49:64-67, (Feb.), 1971.

115. Blankama, L. A., Sacha, H. K., Murray, E. F., and O'Connell, M. J. (Chicago Board Health, Ill.): REPLY BY DR. BLANKSMA AND COLLEAGUES. Letter to the Editor, American Journal of Clinical Pathology, 53: 965-966, (June), 1970.

116. Editorial: LEAD POISONING. Emergency Medicine, 2:39, 42, 45, 46, (Sept.), 1970.

117. Alexiewa, Z., Uschewa, G., and Iwanowa, S. (Inst. Occup. Med., Sofia, Bulgaria): Ueber einige neue kriterien zur Fruehdiagnostik der Bleivergiftungen. (A NEW CRITERION FOR EARLY DIAGNOSIS OF LEAD POISONING). Zeitschrift fuer die Gesamte Hygiene und Ihre Grenzgebiete (Berlin) 18, No. 3:191-192, 1972.

118. Sorkina, N. S., and Evlashko, Yu. P.: K voprosu differentsial' noi diagnostiki nekotorykh professional' nykh anemii. (DIFFERENTIAL DIAGNOSIS OF SEVERAL OCCUPATIONAL ANEMIAS). Klinicheskaya Meditsina (Moscow), 48:83-89, (Mar.), 1970.

119. Harrison, T. R., Adams, R. D., Bennett, I. L., Resnik, W. H., Thorn, G. W., Wintrobe, M. M.: PRINCIPLES OF INTERNAL MEDICINE. McGraw Hill Book Co., New York, 1962, pp. 813-814.

120. Kehoe, R. A.: OCCUPATIONAL LEAD POISONING. Journal of Occupational Medicine, 14:4-5, April-May, 1972.

121. Smith, H. D.: PEDIATRIC LEAD POISONING. Archives of Environmental Health, 8:256-261, (Feb.), 1964.

122. Chisolm, J. J., Jr. and Harrison, H. E.: EXPOSURE OF CHILDREN TO LEAD. Podiatrics 18:943-957, 1956.

123. Goyer, R. A., and Mahaffey, K. R. (Univ. North Carolina, Chapel Hill): SUSCEPTIBILITY TO LEAD TOXICITY. Environmental Health Perspectives Experimental Issue No. 2:73-80, (Oct.), 1972.

124. Freeman, R. (Prince of Wales Hosp., Sydney, Australia): CHRONIC LEAD POISONING IN CHILDREN: A REVIEW OF 90 CHILDREN DIAGNOSED IN SYDNEY, 1948-1967, Australian Paediatric Journal, 5:27-35, (Mar.), 1969.

125. King, B. G. (Bur. Community Environ. Management, PHS, Cincinnati, Oh.): MAXIMUM DAILY INTAKE OF LEAD WITHOUT EXCESSIVE BODY LEAD-BURDEN IN CHILDREN. American Journal of Diseases of Children, 122:337-340, (Oct.), 1971.

126. Maldonado, L., and Ramos, P. H. (Oxtopulco Univ., Mexico): THE KNOWLEDGE OF NATURAL HISTORY OF SATURNISM AS A CLUE TO DIAGNOSIS OF LEAD INTOXICATION. In 16th International Congress on Occupational Health, Tokyo, Japan, September 22-27, 1967 (Pub. 1971), pp. 533-535.

127. Rieke, F. E. (Portland, Ore.): LEAD INTOXICATION IN SHIPBUILDING AND SHIPSCRAPPING 1941-1948. Archives of Environmental Health, 19:521-539, (Oct.), 1969.

128. Stöfen, D. (Montabaur, Germany): THE HEALTH DANGERS OF LEAD IN DRINKING WATER. Zeitschrift für Präventivmedizin, 16:325-332, (July - Aug.), 1971.

129. Chamberlain, M. J. and Massey, P. M. C. (Dudley Road Hosp., Birmingham, England): MILD LEAD POISONING WITH AN EXCESSIVELY HIGH BLOOD LEAD. Notes and Miscellanea, British Journal of Industrial Medicine 29:458-460, (Oct.), 1972.

130. El-Dakhakhny, A. A. and El-Sadik, Y. M. (Alexandria Univ., Egypt): LEAD IN HAIR AMONG EXPOSED WORKERS. American Industrial Hygiene Association Journal, 33:31-34, (Jan.), 1972.

131. Great Britain Department of Employment: HM CHIEF INSPECTOR OF FACTORIES ANNUAL REPORT, 1971, London, Her Majesty's Stationery Office, 1972.

132. Klinge, O. (Univ. Wuerzburg, Germany): Hepatozellulaere Veraenderungen im Leberpunktat bei der Chronischen Bleivergiftung des Menschen (HEPTOCELLULAR CHANGES IN LIVER BIOPSY IN CHRONIC LEAD POISONING IN MAN). Acta Hepto-Splenologica, 16:151-159, (May-June), 1970.

133. U. S. Department of Labor, Occupational Safety and Health Administration: TARGET HEALTH HAZARDS. LEAD. Safety Standards 21:13-17, (Sept.-Oct.), 1972.

134. McAllister, R. G. Jr., Michelakis, A. M., and Sandstead, H. H. (Vanderbilt Univ. Sch. Med., Nashville, Tenn.): PLASMA RENIN ACTIVITY IN CHRONIC PLUMBISM. EFFECT OF TREATMENT. Archives of Internal Medicine, 127:919-923, (May), 1971.

135. Tola, S., Hernberg, S., and Nikkanen, J. (Inst. Occup. Health, Helsinki, Finland): DETECTION OF LEAD EXPOSURE IN SHIPYARDS. In Safety and Health in Shipbuilding and Ship Repairing, International Labour Office, Occupational Safety and Health Series No. 27, 1972, pp. 31-36.

136. Felton, J. S., Kahn, E., Salick, B., VanNatta, F. C., and Whitehouse, M. W. (UCLA Sch. Public Health): HEAVY METAL POISONING: MERCURY AND LEAD. UCLA Conference, Annals of Internal Medicine, 76:779-792, (May), 1972.

137. Poppe, W., and Krauspe, M. (Höchweitz Neurologic Clinic, Döbeln, Germany): Klinische und psychopathologische veränderungen nach Bleiintoxikation. (CLINICAL AND PSYCOPATHOLOGICAL CHANGES FOLLOWING LEAD POISONING). Psychiatria et Neurologia und Medizinische Psychologie (Leipzig), 21:124-130, (Apr.), 1969.

138. Tola, S. (Inst. Occup. Health, Helsinki, Findland): OCCUPATIONAL LEAD EXPOSURE IN FINLAND III. LEAD SCRAP SMELTERS AND SCRAP METAL SHOPS. Work-Environment-Health 11, No. 2:114-117, 1974.

139. Sandstead, H. H., Michelakis, A. M., and Temple, T. E. (Vanderbilt Univ. School of Med., Nashville, Tenn.): LEAD INTOXICATION: ITS EFFECT ON THE RENIN-ALDOSTERONE RESPONSE TO SODIUM DEPRIVATION. Archives of Environmental Health 20:356-363, (Mar.), 1970.

140. Lob, M. (Lausanne Med. Coll., France): saturnisme dans un atelier de montage de batteries. (LEAD POISONING IN A BATTERY ASSEMBLING SHOP). Praxis 58:870-872, (July 8), 1969.

141. Mirando, E. H., and Ranasinghe, L. (Lady Ridgeway Hosp. Children, Colombo, Ceylon): LEAD ENCEPHALOPATHY IN CHILDREN, UNCOMMON CLINICAL ASPECTS. Medical Journal of Australia 2:966-967, (Nov. 21), 1970.

142. Jain, S., O'Brien, B., Fothergill, R., Morgan, H. V., and Geddes, A. M. (East Birmingham Hosp., Birmingham, England): LEAD POISONING PRESENTING AS INFECTIOUS DISEASE. The Practitioner 205:784-786, (Dec.), 1970.

143. Kushner, J. P., Lee, G. R., Wintrobe, M. M., and Cartwright, G. E. (Univ. Utah Coll. Med., Salt Lake City): IDOPATHIC REFRACTORY SIDEROBLASTIC ANEMIA. CLINICAL AND LABORATORY INVESTIGATION OF 17 PATIENTS AND REVIEW OF THE LITERATURE. Medicine (Baltimore), 50:139-159, (May), 1971.

144. Katsunuma, H. (Univ. Tokyo, Japan): HEAVY METAL POISONING. Journal of Japanese Medical Association, 55:1358-1363, 1966.

145. Sanders, L. W., Sr.: TETRAETHYL LEAD INTOXICATION. Archives of Environmental Health, 8:270-277, (Feb.), 1964.

146. Beattie, A. D., Moore, M. R., and Goldberg, A. (Stobhill General Hosp., Glasgow, Scotland): TETRAETHYL-LEAD POISONING. Lancet 2:12-14, (Jul.), 1972.

147. Gratsianskaya, L. N., and Salov'eva, E. N. (Inst. Ind. Hyg. Occup. Dis., Lenningrad, USSR): Priching dlitd' noi invalidnasti pri khronicheskikh Professional 'nykh intoksikatsiyakh. (CAUSES OF PROLONGES INCAPACITATION IN CHRONIC OCCUPATIONAL POISONING). Gigiena Truda i Professional 'nyi Zabolevaniya, 12:10-13, (Aug), 1968.

148. Staik, M., Byczkowska, Z., Szendzikowski, S., and Fiedorczuk, Z. Inst. Occup. Med., Xódź, Poland): ACUTE TETRAETHYL LEAD POISONING. Archiv für Toxikologie 24, No. 4:283-291, 1969.

149. Vurdelja, N., Farago, F., Nikolic, V., and Vučković, S. (Neuropsychiatric Clinic, Novi Sad, Yugoslavia): Klinische Erfahrungen bei Vergiftungen mit Bleitetraäthy) enthaltendem Kraftstoff. (CLINICAL EXPERIENCES WITH INTOXICATIONS OF FUEL CONTAINING LEAD-TETRAETHYL). Folla Facultatis Meditae Univ. Comeniane 5, No. 1: 133-138, 1967.

150. Vučković, S., Šovljanski, R., and Balley, R. (Neuropsych Clin., Novi Sad, Yugoslavia): Professionalna Otrovanja tetraetilolovom u sap Vojrodini. (OCCUPATIONAL TETRAETHYL LEAD POISONING IN SAP VDIVODINA). Arhiv za Higijenu Rada i Toksikologiju (Zagreb) 21: 265-269, 1970.

151. Devoto, G. (Univ. Cagliari, Italy): (DETERMINATION OF LEAD IN URINE AND BLOOD BY ATOMIC ABSORPTION SPECTROSCOPY). Bolletino della società Italiana di Biologia Sperimentale 44, No. 5:421-423, 1968.

152. Scarlato, G., Smirne, S., and Poloni, A. E. (Univ. Milan, Italy): L'encefalopatia saturnina acuta dell' adulto. Descrizione anatomo clinica di un caso. (ACUTE ENCEPHALOPATHY DUE TO LEAD POISONING IN THE ADULT: CLINICAL AND PATHO ANATOMICAL FINDINGS IN A CASE). Acta Neurologica (Naples) 24, No. 4:578-580, 1969.

153. Maljkouic, I.: Slucaj kronicnog profesional nog otrovanja olovnim stearatom i karbonatom. (A CASE OF CHRONIC OCCUPATIONAL POISONING BY LEAD STEARATE AND LEAD CARBONATE). Sigurnost a pogonu (Zagreb, Yugoslavia), 13:123-124, (May), 1971. (From Occupational Safety and Health Abstracts 10, No. 2:106, 1972.

154. Caille, M. P., Meyrand, D., and Andlauer, P. (Regional Med. Insp. of Labor, Rhone-Alpes, France): Formes aigues de saturnisme par stearate de plomb. (ACUTE FORMS OF LEAD POISONING BY LEAD STEARATE). Bohhives des Maladies Professionelles de Medecine du Travail et de Securite Sociale 32:573-575, (Sept.), 1971.

155. Braun, W., and Cutjahr, L. (Neurol. Div., Nordstadt Hosp., Hanover, W. Germany): Bleienzephalopathie mit Hirndrucksteigerung bei Erwachsenem. (LEAD ENCEPHALOPATHY WITH INTRACRANIAL PRESSURE IN ADULTS). Zentralblatt fuer Neurochirurgie (Leipzig) 32, No. 3-4:167-172, 1971.

156. Buckharovich, M. N., Speranskii, N. N., Zakharov, I. Ya., and Malitsku, A. F., Zabolevaniya kozhiu rabotnikov tsekha proizvodstva tsirkonata titanata svintsa (TsTS-23, TsTS-24), (SKIN DISEASES IN WORKERS OF A DEPARTMENT ENGAGED IN THE PRODUCTION OF LEAD TITANATE ZIRCONATE (LTZ-23, LTZ-24).) Gigiena Truda i Professional'nye Zabolevaniya 16 No. 6:35-37, 1972.

157. Packham, R. F. (Water Res. Assoc., England): THE LEACHING OF TOXIC STABILIZERS FROM UNPLASTICIZED PVC WATER PIPE: PART I - A CRITICAL STUDY OF LABORATORY TEST PROCEDURES; PART II - A SURVEY OF LEAD LEVELS IN uPVC DISTRIBUTION SYSTEMS; PART III - THE MEASUREMENTS OF EXTRACTABLE LEAD IN uPVC PIPES. Water Treatment and Examination 20, Part 2:108-124; Part 3: 144-151, 152-164, 1971.

158. Gambini, G., and Farina, G. (Univ. Milan, Italy): Su di un caso di aturnismo associato a sclerosi laterale amiotrofica. (ON A CASE OF SATURNISM ASSOCIATED WITH AMYOTROPHIC LATERAL SCLEROSIS). Medicina del Lavor, 59:599-603, (Oct.), 1968.

159. Gattolet, M., Chalabreysse, J., and Ardiauer, P. (Ind. Med. Inspection, Rhone-Alps, France): Nuisance des sels de plomb dans l'industrie des matieres plastiques, (NOXIOUSNESS OF LEAD SALTS IN THE PLASTIC INDUSTRY). Archives des maladies Professionnelles de Medecine du Travail et de Securite Sociale 35:685, (June), 1974.

160. Faritskaya, L. A., and Zdorovtseva, T. N. (Kiev Inst. Ind. Hyg. Occup. Dis., USSR): Progressiruyushchee techenie svintsovogo polinevrita. (PROGRESSIVE COURSE OF LEAD POLYNEURITIS). Vrachebnoe Delo 7:147-149, (July), 1968.

161. Bister, F., and Franke, W. (Lower Saxony Prov. Dept. Ind. Med. Occup. Hyg., Hanover, Germany): Bleiausschwemmung durch Mosatil noch Auftreten akuten Bleisymptome bei 16 Farbspritzern. (PROMOTION OF LEAD EXCRETION BY MOSATIL IN 16 PAINT SPRAYERS WITH ACUTE SYMPTOMS OF LEAD POISONING). Arbeitsmedizin Sozialmedizin Arbeitshygiene 5, No. 8:225-226, 1970.

162. Baghdassanian, S. A. (John Hopkins Univ. School Med., Baltimore, Md.): OPTIC NEUROPATHY DUE TO LEAD POISONING REPORT OF A CASE. Archives of Ophthalmology, 80:721-723, (Dec.), 1968.

163. Tomza, J. (Zarach Hosp, Poland): Krwotoczny nieżyt ż ołądka w przebiegu przewlekłego zatrucia o owiem. (HEMORRHAGIC GASTRITIS IN THE COURSE OF CHRONIC LEAD POISONING). Wiadomosci Le karskie, 19:491-493, (Mar. 15), 1966.

164. Barbacki, M. (Ind. Hyg. Consultation Div., Krakow, Poland): Przypadek przewlektej ołowicy z nietypowymi objawami neurologicznymi. (A CASE OF CHRONIC LEAD POISONING WITH ATYPICAL NEUROLOGICAL MANIFESTATIONS). Wiadomosci Lekarskie, 22:2211-2215, (Dec. 15), 1969.

165. Sklenský, B. (Univ. J. E. Purkyne, Brno, Chechoslovakia): Subakutni otravy olovem. (SUBACUTE LEAD POISONING). Vnitřni Lékařstuí 15, No. 1: 89-92, 1969.

166. U. S. Public Health Service, Center for Disease Control: EPIDEMOLOGICAL NOTES AND REPORTS. LEAD POISONING - VIRGINIA. Morbidity and Mortality Weekly Report, 24:150, 155, (Apr. 26), 1975.

167. Philbert, M., Valcke, J. -C., Bestel, B., Poisson, M., Rancurel, G., Buge, A. and Moreau, L. (Inst. Ind. Hyg. Occup. Med., Paris, France): Paralysies saturnines d' evolution generalissee chez un chalumiste. (LEAD INDUCED PARALYSIS OF GENERALIZED DEVELOPMENT IN A WELDER). Archives des Maladies Professionnelles de Medecine du Travail et de Securite Sociale, 35:739-748, (July-Aug.), 1974.

168. Linch, A. L., Weist, E. G., and Carter, M. D. (duPont Co., New Jersey): EVALUATION OF TETRAALKYL LEAD EXPOSURE BY PERSONNEL MONITOR SURVEYS. American Industrial Hygiene Association Journal 30: Conference Program, p. 142, (Mar. Apr.), 1969.

169. Boehme, D. H. (VA Hosp., East Orange, N. J.): MYELOPATHIA SATURNINA, REPORT OF A CASE. Acta Neuropathologica (Berlin), 18:356-360, 1971.

170. Saita, G., and Lussana, S. (Beogamo Hosp., Italy): Intossicazione da piombo in portatrice di emazie fabiche. (LEAD POISONING IN A PATIENT WITH FAVISM). Medicina del Lavoro, 62:22-27, (Jan.), 1971.

171. Girard, R., Porte, J., Rigaut, P. and Tolot, F. (Hosp. Edouard-Herriot, Lyons, France): Exposition saturnine méconnue dans les travaux d'entretien d'un tunnel routier. (UNRECOGNIZED LEAD POISONING IN THE MAINTENANCE WORK OF A HIGHWAY TUNNEL). Archives des Maladies Professionelles de Médecine du Travail et de Sécurité Sociale, 31:318-319, (June), 1970.

172. Sanai, G. H., Jiai, N., Ghlami, A., and Ghasemi, A. (Univ. Teheran, Iran): INVESTIGATION OF LEAD INTOXICATION IN TEHERAN TILE MANUFACTURING WORKERS. Industrial Health, 11:197-201, (Dec.), 1973.

173. Chianura, G. (Inst. Radiol., Radium L. Galvani, Italy): Alterazoni radiologiche parotides da avvelen amento Cronico da Piombo. (RADIOLOGICAL PAROTID CHANGES FROM CHRONIC LEAD POISONING). Bullettino delle Scienze Mediche 144, No. 2:125-130, 1972.

174. Alberti, S., Salimei, E., and Cioppi, E. (Inst. Ind. Med., ENPI, Ancona, Italy): La prevenzione del reschio da prombo in un ambiente artigianale. (PREVENTION OF LEAD HAZARDS IN AN ARTISAN SHOP). Securitos 57, No. 2-3: 147-159, 1972.

175. Hassman, P. (Occup. Dis. Clinic, Königgrätz, Czechoslovakia): Otravy oloven při výrobe automobilových karoserii. (LEAD POISONING IN MOTORCAR BODY PRODUCTION). Prakticky Lekar 50, No. 1:7-10, 1970.

176. Kühnen, G. (Dust Res. Inst., Trade Unions, Bonn, Germany): LEAD DUST CONCENTRATION IN THE INITIAL STAGE OF AUTOMOBILE BODY BUILDING. Staub, Reinhaltung der Luft in English, 29:10-15, (Feb.), 1969.

177. Martenka, E., and Mrozek, J. (Prov. Hosp. Nervous Diseases, Swieciu, Poland): Przypadek zaburzen psychicznch w przehiegu prezewlek/ego zatrucia o/owiem. (A CASE OF MENTAL DISORDERS IN THE COURSE OF CHRONIC LEAD POISONING). Psyshiatria Polska, 2:675-678, (Nov.-Dec.), 1968.

178. Alétru, A.: Evolution de la médecine du travail dans L'imprimerie. (EVOLUTION OF OCCUPATIONAL MEDICINE IN THE PRINTING INDUSTRY). Archives des Maladies Protesionelles de Médecine du Travail et de Sécurité Sociale, 29:220-221, (Apr.-May), 1968.

179. Girard, R., Boubrier, R., Brennon, J. P., and Totot, F. (Hosp. Edouard-Herriot, Lyons, France): saturnisme et vieilles statues. (LEAD POISONING AND OLD STATUES). Archives des Maladies Protessionnelles de Medicine du Travail et de Securite Sociale, 31:309-311, (June), 1970.

180. St. George, I. M. (Dunedin Hosp., New Zealand): TWO MEN WITH LEAD POISONING. New Zealand Medical Journal, 71:294-297, (May), 1970.

181. Chisolm, J. J. (Baltimore City Hosp., Md.): CASE FINDINGS: USES AND LIMITATIONS. In Environmental Sciences, Fogarty International Center and Human Health, D. H. K. Lee, Ed. New York, Academic Press, Inc., 1972, pp. 209-227.

182. Morgan, J. M., Oh, S. J., and Linn, J. E., Jr., (Univ. Alabama Sch. Med., Birmingham, Alabama): LEAD NEUROPATHY IN ALCOHOLS. Alabama Journal of Medical Sciences 8:67-74, (Jan.), 1971.

183. University of Alabama Medical Center, Medical Grand Rounds: LEAD POISONING. Southern Medical Journal, 65:278-288, (Mar.), 1972.

184. Patterson, M., and Jernigan, W. C. T. (Med. Center, Columbus, Ga.): LEAD INTOXICATION FROM "MOONSHINE." GP:126-131, (Oct.), 1969.

185. Editorial (Kinenborg, J. R.): SATURNINE GOUT - A MOONSHINE MALADY. New England Journal of Medicine, 280:1238-1239, (May 29), 1969.

186. Owen, C., Jr., Dodson, W. H., and Hammack, W. J., Eds. (Univ Alabama, Birmingham): MEDICAL GRAND ROUNDS FROM THE UNIVERSITY OF ALABAMA MEDICAL CENTER. Southern Medical Journal, 60:177-184, (Feb.), 1967.

187. Cheatham, J. S., and Chobot, E. F., Jr. (Baroness Erlanger Hosp., Chattanooga, Tenn.): THE CLINICAL DIAGNOSIS AND TREATMENT OF LEAD ENCEPHALOPATHY. Southern Medical Journal, 61:529-531, (May), 1968.

188. Crawford, O., Jr., Dodson, W. H., and Hammack, W. J., Eds.: MEDICAL GRAND ROUNDS FROM THE UNIVERSITY OF ALABAMA MEDICAL CENTER. Southern Medical Journal, 60:44-50, (Jan.), 1971.

189. Wülfert, K. (Yrkeshygienisk Inst., Oslo, Norway): Et eiendommelig blyforgiftning-stilfelle. (AN UNUSUAL CASE OF LEAD POISONING). Nordisk Hygienisk Tidskrift 49, No. 2:39-43, 1968.

190. Crutcher, J. C. (Emory Univ. School of Med., Atlanta, Ga.): LEAD INTOXICATION AND ALCOHOLISM: A DIAGNOSTIC DILEMMA. Journal of the Medical Association of Georgia, 56:1-4, (Jan.), 1967.

191. Ball, G. V. (Univ. Alabama, Birmingham): TWO EPIDEMICS OF GOUT. Bulletin of the History of Medicine, 45:401-408, (Sept.-Oct.), 1971.

192. Choisy, H., Dufour, M., Lacour, F., Miramcourt, G., Morice, M. T., and Potron, G. (Hosp. Center Univ. Reims, France): Saturnisme et cruches plomb feres. (LEAD POISONING AND LEAD CONTAMINATED BEVERAGE PITCHERS). Annales Medicale de Reims, 8:315-319, 1971.

193. Boudin, C., Pepin, B., Vernant, J. C., and Gautier, B. (Hosp. Salpêtrière, Paris, France): Polyradiculonévrite saturnine généralisée au course d'une intoxication collective par le plomb. (GENERALIZED SATURINE POLYRADICULONEURITIS IN THE COURSE OF A COLLECTIVE LEAD POISONING). Annales de Medecine Interne (Paris), 121:363-366, (Mar.), 1970.

194. Jaulmes, P., and Hamelle, G. (Coll. Pharm., Montpellier, France): Deux cas d'intoxication saturnine due a l'emploi de récipients en tole plombée pour le transport du vin. (2 CASES OF LEAD POISONING DUE TO THE USE OF LEAD PLATED CONTAINERS FOR THE TRANSPORT OF WINE). Medecine Legale et Dommage Corporel (Paris), 1:218, (Apr.), 1968.

195. Luard, J. P.: Le saturnisme du au vin. (LEAD POISONING ATTRIBUTABLE TO WINE). Thesis, Paris, 1967.

196. Clark, K. G. A. (Kings Coll. Hosp. Med. Sch., London, England): LEAD-GLAZED EARTHENWARE. Letter to the Editor. Lancet, 2:662-663, (Sept.), 1972.

197. Walls, A. D. F. (St. Luke's Hosp., Guildford, Surrey, England): HOME-MADE CIDER: SOURCE OF LEAD POISONING. British Medical Journal, 1: 98, (Jan. 11), 1969.

198. Danjou, P. (Marine Personnel Physician, France): A propos d'une intoxication collective alimentaire par le plomb a bord d'un petrolier. (COLLECTIVE ALIMENTARY LEAD POISONING ABOARD A TANKER). Revue des Corps de Sante Armees 13, No. 3:255-263, 1972.

199. Osterud, H. T., Tufts, E., and Holmes, M. A. (Univ. Oregon Med. Sch., Portland): PLUMBISM AT THE GREEN PARROT GOAT FARM. Clinical Toxicology 6, No. 1:1-7, 1973.

200. Baumann, H. W., Swanson, M., Bossatti, E. B., and Holmes, M. A., (Univ. Oregon Med. Sch. Hosp. and Clinics, Portland): EPIDEMOLOGIC NOTES AND REPORTS. LEAD POISONING - OREGON. Morbidity and Mortality, 20:199-200, (June 5), 1971.

201. Biro, L., Szanyi, E., and Soos, G. (Natl. Ind. Health Dept., Budapest, Hungary): Acut intermittalo porphyriat utanzo, alimentaris eredetu alommergezes. (ALIMENTARY LEAD POISONING, SIMULATING ACUTE INTERMITTENT PORPHYRIA). Orvosi Hetilap, 112:2349-2351, (Sept.), 1971.

202. Bozhinov, S., Georgiev, Iv., and Makedonska, D. (Katedra Nevial., Sofia, Bulgaria): (ON SOME ASPECTS OF THE CLINICAL AND THE PATHOANATOMICAL PICTURE OF SATURNINE POLYNEURITIS). Nevrol., Psikhiat, Nevrokhir. 5, No. 5:345-353, 1966.

203. Cvetanov, V., Kotevski, L., and Marković, N.: Trovanje olovom iz zemljanog posuda u SR makedoniji. (LEAD POISONING FROM EARTHENWARE DISHES AND POTS IN THE MACEDONIAN SR). From Yugoslav Association on Occupational Health and Trepča Works. International Symposium on Lead Poisoning. Ahiv za Higijenu Rada i Toksikologiju 20, No. 4: 457-458, 1969.

204. Sverdlor, S. L. (Novozybkor Mun. Hosp., Bryansk Distr., USSR): Ob oshibochnykh laparotomiyakh pri svintsovoi kolike u bol'nykh bytovym saturnizmom. (ERRONEOUS LAPAROTOMY IN LEAD COLIC IN PATIENTS WITH DOMESTIC SATURNISM). Klinicheskaya Meditsina (Moscow), 47:141-143, (May), 1969.

205. Cambell, E. E., Miller, H. M., and Biere, M. R. (Sci. Lab., Los Alamos, N. Mexico): THE CONCEALED LEAD IN POTTERY FROM TONALA. El plomo oculto en la cerámica de tonala. Salud Publica de Mexico, 8:905-908, (Nov.-Dec.), 1966.

206. Dickinson, L., Reichert, E. L., Ho, K. C. S., Rivers, J. B., and Kominami, N. (Univ. Hawaii, Honolulu): LEAD POISONING IN A FAMILY DUE TO COCKTAIL GLASSES. American Journal of Medicine, 52:391-394, (Mar.), 1972.

207. Fromke, V. L., Lee, M. Y., and Watson, C. J. (Northwestern Hosp., Minneapolis, Minn.): PORPHYRIN METABOLISM DURING VERSENATE THERAPY OF LEAD POISONING. INTOXICATION FROM AN UNUSUAL SOURCE. Annals of Internal Medicine, 70:1007-1012, (May), 1969.

208. U. S. Food and Drug Administration: REGIONAL WARNING TO CONSUMERS NOTES HIGH LEAD LEVELS IN DINNERWARE ITEMS. FDA Papers, 4:35, (June), 1971.

209. Rosenbaum, D. L., Silverman, A. G., and Okun, R. (Cedar-Sinai Med. Center, Los Angeles, Ca.): PLUMBISM FROM THE SALT SHAKER. Abstracts: The American Academy of Clinical Toxicology, San Diego, California, July-August, 1973. Clinical Toxicology 7, No. 2:208, 1974.

210. Green, V. A., Wise, G. W., and Small, N. W. (The Children's Mercy Hosp., Kansas City, Mo.): LEAD SURVEY OF SELECTED CHILDREN IN KANSAS CITY AND SOME UNUSUAL CASES. Clinical Toxicology 6, No. 1:29-37, 1973.

211. Beattie, A. D., Dogg, J. H., Goldberg, A., Wang, I., and Ronald, J. (Stobhill Hosp., Glasgow, Scotland): LEAD POISONING IN RURAL SCOTLAND. British Medical Journal, 2:488-491, (May), 1972.

212. Williams, M. K.: LEAD POISONING: AN UNUSUAL COMPLICATION OF CYSTITIS. Correspondence. Lancet, 2:480, (Sept. 2), 1972.

213. Capellini, A., and Gambini, G. (Univ. Milan, Italy): Due casi di saturnismo extra-professionale. (TWO CASES OF NON-OCCUPATIONAL LEAD POISONING). Medicina del Lavoro, 62:391-397, (Aug.-Sept.), 1971.

214. Sundaravalli, N., Sundaram, V. M., and Balagopal, R. V. (Inst. Child Health Hosp. Children, Madros, India): LEAD ENCEPHALOPATHY: REPORT OF A CASE. Indian Journal of Pediatrics, 39:199-202, (June), 1973.

215. Baizakova, S. S., Chupakhina, Z. G., and Zharova, V. D. (Rep. Clin. Hosp., Kirgiz, USSR): Sluchai tyazhe; ogo svintsovogo otravleniya v bytu. (A CASE OF ACUTE LEAD POISONING IN EVERYDAY LIFE). Sovetskoe Zdravookhranenie Kirgizii, 2:63-64, (Mar.-Apr.), 1970.

216. Shellshear, I. D. (Christchurch Public Hosp., New Zealand): LEAD POISONING IN CHILDHOOD: A CASE REPORT WITH ENVIRONMENTAL IMPLICATIONS. New Zealand Medical Journal, 78:251-254, (Sept.), 1973.

217. Cohen, C. J., Bowers, G. N., and Lepow, M. L. (Univ. Connecticut Health Cent. Sch. Med., Farmington, Ct.): EPIDEMIOLOGY OF LEAD POISONING, A COMPARISON BETWEEN URBAN AND RURAL CHILDREN. Journal of the American Medical Association, 226:1430-1433, (Dec.), 1973.

218. Fejerman, N., Gimenez, E. R., Vallejo, N. E., and Medina, C. S. (Children's Hosp., Buenos Aires, Argentina): LENNOX'S SYNDROME AND LEAD INTOXICATION. Pediatrics, 52:227-234, (Aug.), 1973.

219. Russo, R. M., Laude, T., Rajkumar, S., and Allen, J. E. (State Univ. of N. Y. Dowstate Med. Center, Brooklyn): INCIDENCE OF PLUMBISM AND PICA IN ACCIDENTALLY POISONED CHILDREN. New York State Journal of Medicine, 73:1881-1885, (July), 1973.

220. De La Burde, B., and Laupus, W. E. (Med. Coll. Virginia, Richmond): SUBCLINICAL LEAD POISONING IN A GROUP OF CHILDREN. Virginia Medical Monthly, 100:623-628, (July), 1973.

221. Alexander, F. W., and Delves, H. T. (Hosp. Sick Children, London, England): DEATHS FROM ACUTE LEAD POISONING. Archives of Disease in Childhood 47:446-448, (June), 1972.

222. King, B. G., Schaplowsky, A. F., and McCabe, E. B. (Bur. Community Environ. Management, U. S. Public Health Serv., Cincinnati, Oh.): OCCUPATIONAL HEALTH AND CHILD LEAD POISONING: MUTUAL INTERESTS AND SPECIAL PROBLEMS. American Journal of Public Health, 62:1056-1059, (Aug.), 1972.

223. Chisolm, J. J., Jr., (Baltimore City Hospitals, Md.): MANAGEMENT OF INCREASED LEAD ABSORPTION AND LEAD POISONING IN CHILDREN. New England Journal of Medicine, 289:1016-1018, (Nov. 8), 1973.

224. American Academy of Pediatrics, Subcommittee on Accidental Poisoning (Alpert, J. J., Chairman): PREVENTION, DIAGNOSIS, AND TREATMENT OF LEAD POISONING IN CHILDHOOD. Pediatrics, 44:291-298, (Aug.), 1969.

225. Bicknell, J. (Queen Mary's Hosp. for Children, Carshalton, Surrey, England): SELECTIVE PICA AND LEAD POISONING IN A SEVERELY SUB-NORMAL CHILD. Journal of Mental Deficiency Research, 11:278-281, (Dec.), 1967.

226. Ames, A. C., and Swift, P. N. (Lewisham Hosp., London, England): LEAD POISONING IN BLIND CHILDREN. British Medical Journal, 3:152-153, (July 20), 1968.

227. Cohen, B. L., Harper, D. L., and Neal, W. (Doctors Hosp., Columbus, Oh.): CHRONIC LEAD POISONING IN CHILDREN: A CASE REPORT AND REVIEW OF THE LITERATURE. Journal of the American Osteopathic Association, 67:1148-1152, (June), 1968.

228. Freeman, R. (Prince of Wales Hosp., Sydney Hosp., Sydney, Australia): CHRONIC LEAD POISONING IN CHILDREN: A REVIEW OF 90 CHILDREN DIAGNOSED IN SYDNEY, 1948-1967. 1. EPIDEMIOLOGICAL ASPECTS. 2. CLINICAL FEATURES AND INVESTIGATIONS. Medical Journal of Australia, 1:640-647; 648, 651, (Mar. 28), 1970.

229. Graef, J. W., Kopito, L., and Shwashman, H. (Children's Hosp. Med. Center, Boston, Mass.): LEAD INTOXICATION IN CHILDREN: DIAGNOSIS AND TREATMENT. Postgraduate Medicine, 50:133-138, (Dec.), 1971.

230. Gilsinn, J., Nocks, B., and Clark, E. (Natl. Bur. Standards, Washington, D. C.): THE NATURE OF THE LEAD PAINT POISONING HAZARD. U. S. National Bureau of Standards Report 10:499, 1971.

231. Aronow, A. M. (Los Angeles, Calif.): LEAD POISON IN PUTTY. (Letters) Science, 166:552, (Oct. 31), 1969.

232. Lead Industries Association, Inc.: FACTS ABOUT LEAD AND PEDIATRICS. New York, N. Y., 1971.

233. Gelli, G.: Intossicazione da piombo nel bambino. (LEAD INTOXICATION IN CHILDREN). Minerva Pediatrica, 19:1570-1573, (Aug. 18), 1967.

234. Veliev, B. A. (Med. Soc. Kazakh SSR, Chimkent): Svintsovaya intoksikatsiya. (LEAD POISONING). Zdravookhranenie Kazakhstana 7:13-14, 1967.

235. Joshua, G. E. (Christian Med. Coll., Vellore, S. India): CHRONIC LEAD POISONING: RECURRENT ENCEPHALOPATHY IN A CHILD. Indian Pediatrics, 6:329-337, (May), 1969.

236. Joshua, G. E., Ratnaike, N., and Benjamin, V. (Christ. Med. Coll., Vellore, Madras, India): LEAD POISONING IN A FAMILY OF 18 MEMBERS IN VELLORE TOWN. Indian J. Med. Res. 59, No. 9: 1496-1507, 1971.

237. Carella, A. (Univ. Rome, Italy): Del saturnismo in lattanti per l'uso, da parte delle nutrici di paracapezzoli di piombo. (ON LEAD POISONING IN INFANTS DUE TO LEAD NIPPLE SHIELDS USED BY WET NURSES). Nuovi Annali d'Igiene e Microbiologia, 18:445-455, (Nov.-Dec.), 1967.

238. Kombe, A. (Muhimbili Hosp., Dares Salaam, Tanzania, E. Africa): LEAD POISONING IN AN AFRICAN INFANT. (A CASE REPORT). East African Medical Journal, 49:621-623, (Aug.), 1972.

239. Hankin, L., Heichel, G. H., and Batsford, R. A. (Connecticut Agric. Expt. Sta., New Haven): LEAD POISONING FROM COLORED PRINTING INKS, A RISK FOR MAGAZINE CHEWERS. Clinical Pediatrics, 12:654-655, (Nov.), 1973.

240. Wolf, M. D. (Children's Hosp. District of Columbia, Washington): LEAD POISONING FROM RESTORATION OF OLD HOMES. Letters Journal of the American Medical Association, 225:175-176, (July), 1973.

241. Gleason, M. N., Gosselin, R. E., Hodge, H. C., and Smith, R. P.: CLINICAL TOXICOLOGY OF COMMERCIAL PRODUCTS. The Williams & Wilkins Co., Baltimore, 1969, pp. 137-140.

242. Kehoe, R. A.: INDUSTRIAL LEAD POISONING. Editors: Patty, F. A., Fassett, D. W., and Irish, D. D.: INDUSTRIAL HYGIENE AND TOXICOLOGY VOLUME II. Interscience Publishers, New York, 1967, pp. 941-980.